The Journal of Bone and Joint Surgery

The Official Publication of
The American Orthopaedic Association
The British Orthopaedic Association
The American Academy of Orthopaedic Surgeons
The Australian Orthopaedic Association
The Canadian Orthopaedic Association
American Society for Surgery of the Hand
New Zealand Orthopaedic Association
South African Orthopaedic Association
Western Orthopedic Association

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The Journal of Bone and Joint Surgery*

American Volume

TABLE OF CONTENTS

December 1960

PAGE

Injuries to the Medial Ligaments of the Ankle. Result Study.
By *O. Sherwin Staples, M.D.*, Hanover, New Hampshire 1287

Arthrodesis of the Ankle with Particular Reference to the Gallie Procedure. A Review of Fifty Cases.
By *J. C. Kennedy, M.D., F.R.C.S. (C)*, London, Ontario, Canada 1308

Roentgen Aspects of Injuries to the Pisiform Bone and Pisotriquetral Joint.
By *Anthony Vasilas, M.D., R. Vincent Grieco, M.D.*, and *Noel F. Bartone, M.D.*, New York, N. Y. 1317

Unusual Case of Multiple Abnormalities of Bone with Malignant Degeneration.
By *Albert G. Smith, M.D., Ralph W. Coonrad, M.D., Delos W. Boyer, M.D.*, and *Everett I. Bugg, Jr., M.D.*, Durham, North Carolina 1329

Further Studies on Experimental Spondylitis and Intercorporeal Fusion of the Spine.
By *C. Roger Sullivan, M.D.*, and *Frank E. McCaslin, Jr., M.D.*, Rochester, Minnesota. 1339

A Study of Strontium^{85} Metabolism in the Normal and in the Castrated Mouse.
By *Lee J. Cordrey, M.D., James H. Christie, M.D., William J. MacIntyre, Ph.D.*, and *Rufus Palmer, B.S.*, Cleveland, Ohio 1349

The Treatment of Profundus Division by Free Tendon Graft.
By *R. Guy Pulvertaft, F.R.C.S.*, Derby, England 1363

Adduction Injury of the Knee with Rupture of the Common Peroneal Nerve. Report of a Case.
By *Max M. Novich, M.D.*, Newark, New Jersey 1372

Congenital Malformation of the Atlanto-Axial Joint with Dislocation.
By *Colonel Earl W. Brannon*, Medical Corps, United States Air Force 1377

Growth Following Transepiphyseal Bone Grafts. An Experimental Study to Explain Continued Growth Following Certain Fusion Operations.
By *J. T. H. Johnson, M.D.*, Baltimore, Maryland, and *Wayne O. Southwick, M.D.*, New Haven, Connecticut 1381

(Continued on page 3)

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TABLE OF CONTENTS

(*Continued from page 1*)

PAGE

Bone Growth after Spine Fusion. A Clinical Survey.
By *J. T. H. Johnson, M.D.*, Baltimore, Maryland, and *Wayne O. Southwick, M.D.*, New Haven, Connecticut 1396

The Problem of the Primary Curve.
By *John R. Cobb, M.D.*, New York, N. Y. 1413

The Effect of a Tibial-Tubercle Transplant in Children on the Growth of the Upper Tibial Epiphysis.
By *J. William Fielding, M.D., William A. Liebler, M.D.*, and *A. Tambakis, M.D.*, New York, N. Y. 1426

A Study of the Residual Nitrogen in Bone Following Extraction with Ethylenediamine.
By *Lieutenant Commander Thomas E. Wheeler*, Medical Service Corps, United States Navy, and *Captain George W. Hyatt*, Medical Corps, United States Navy 1435

Proceedings:

The American Orthopaedic Association 1441

Major General Norman Thomas Kirk 1450

Book Reviews 1453

The Orthopaedic Research Society 1457

Index to Volume 42-A

Authors 1458

Subjects 1465

List of Advertisers ..... Adv. 58

Addresses of Contributors ..... Cover III

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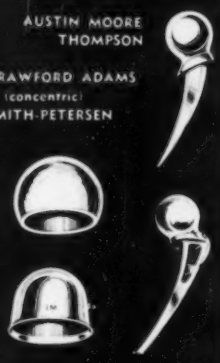

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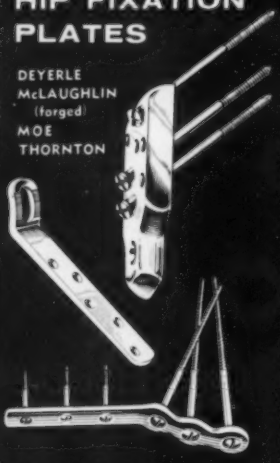

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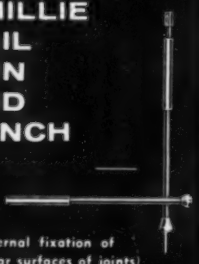

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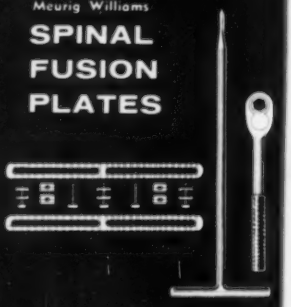

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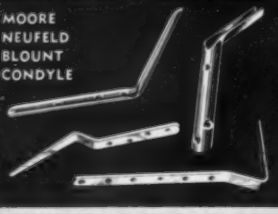

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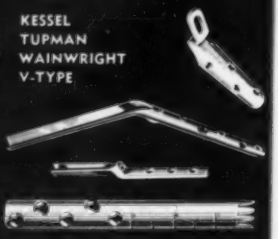

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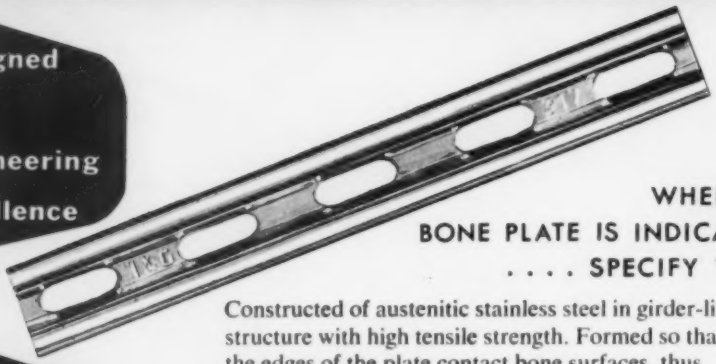

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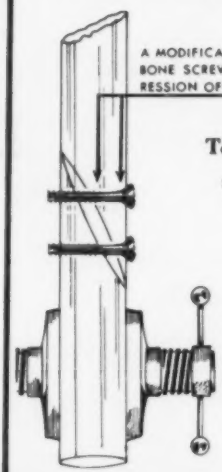

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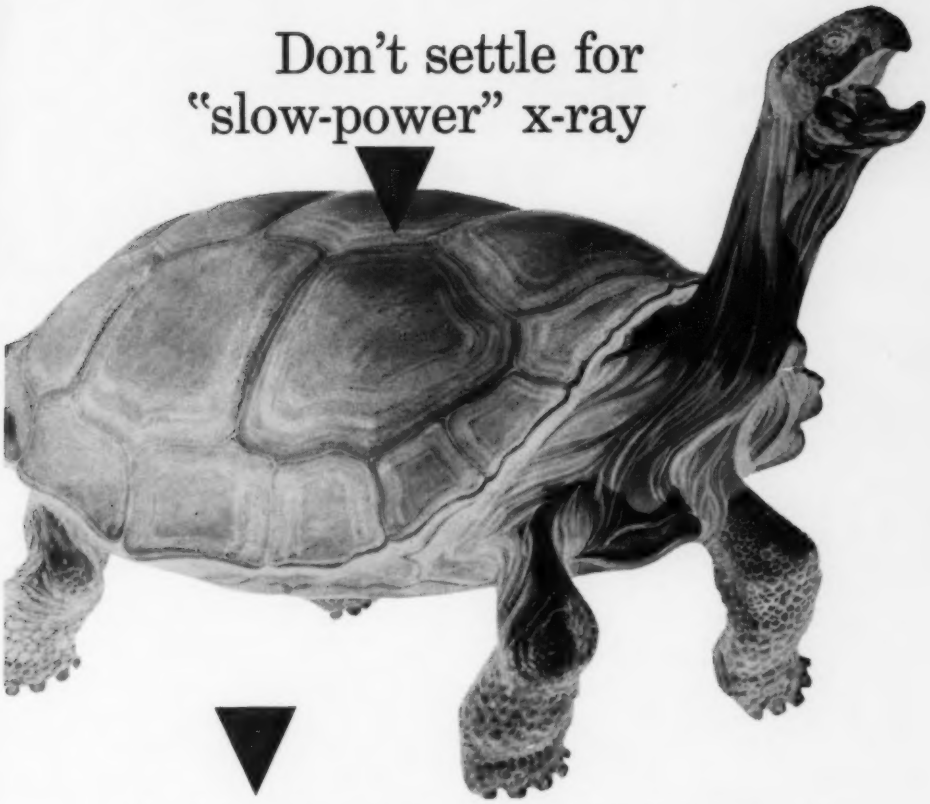

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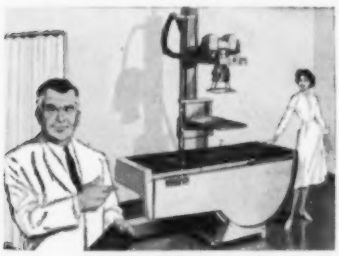

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Soma Compound is an entirely new, totally different analgesic combination that contains three drugs. First, Soma: a new type of analgesic that has proved to be highly effective in relieving both pain and tension.* Second, phenacetin: a "standard" analgesic and antipyretic. Third, caffeine: a safe, mild stimulant for elevation of mood. As a result, the patient gets more complete relief than he does with other analgesics. Soma Compound is nonnarcotic and nonaddicting. It reduces pain perception without impairing the natural defense reflexes.*

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in *TENOSYNOVITIS*— often frees "locked" tendons without need for surgery

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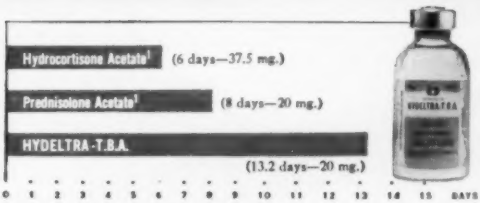
Anti-inflammatory effect lasts longer than that provided by any other steroid ester


Hydrocortisone Acetate[1] (6 days—37.5 mg.)
Prednisolone Acetate[1] (8 days—20 mg.)
HYDELTRA-T.B.A. (13.2 days—20 mg.)
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Dosage: the usual intra-articular, intra-bursal or soft tissue dose ranges from 20 to 30 mg. depending on location and extent of pathology.

Supplied: Suspension 'HYDELTRA'-T.B.A.—20 mg./cc. of prednisolone *tertiary*-butylacetate, in 5-cc. vials.

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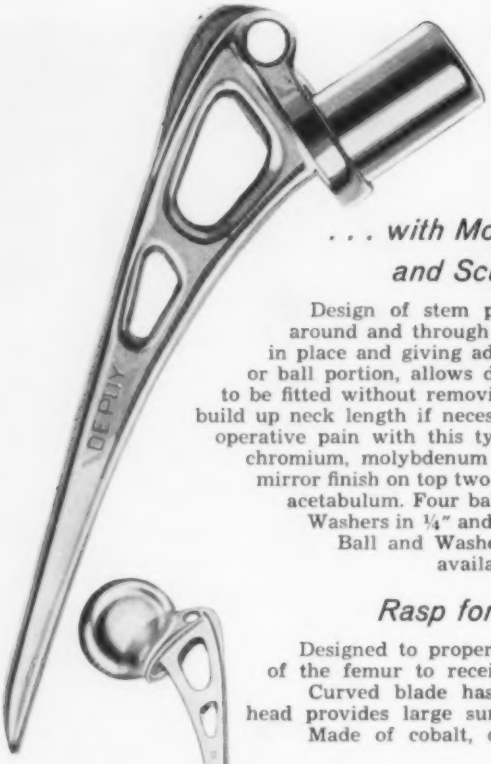
De Puy

HIP PROSTHESIS



DePuy Hip Prosthesis
No. 775

. . . with Moore Self-Locking Stem and Scuderi Detachable Head

Design of stem permits bony growth to occur around and through the openings, locking it firmly in place and giving added strength. Detachable head, or ball portion, allows different sizes of femoral heads to be fitted without removing entire unit. Use washers to build up neck length if necessary. Doctors report less post-operative pain with this type prosthesis. Made of cobalt, chromium, molybdenum alloy with satin finish except mirror finish on top two-thirds of ball where it contacts acetabulum. Four ball sizes: 1¾", 1⅞", 2" and 2⅛". Washers in ¼" and ⅛" thicknesses. No. 775 (Stem, Ball and Washer). Moore Hip Prosthesis also available in one-piece unit, No. 826.

DePuy Hip Prosthesis
No. 826

Rasp for Moore Hip Prosthesis

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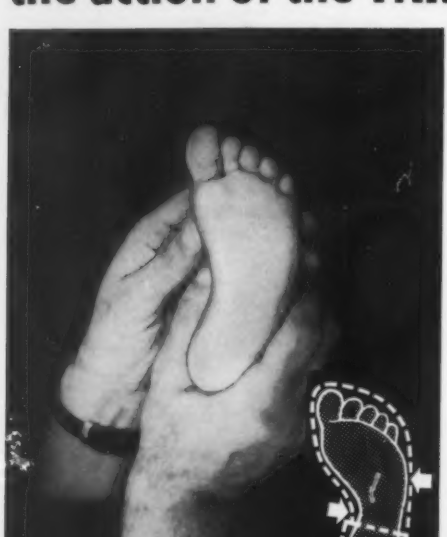
the action of the TARSO SUPINATOR SHOE



. . . swings the hind-foot inward, which induces adduction of the fore-foot, elevates the longitudinal arch, and levels the ankle joint.

When the hind-foot is held in a position of inward rotation, the talo-scaphoid and cuneiform-scaphoid joints will remain locked under weight bearing. The arch will not collapse and the ankle will not roll inward.

Correction is accomplished by the specially shaped last. Medially wedged and extended heels promote better weight distribution. Medially extended counters are used for utmost control.

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Tarso Shoes are ready-made and are available on prescription from dealers in most cities. Write for catalog and name of nearest dealer.

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FOR FLAT FEET

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for muscle relaxation *plus* analgesia

Prescribe PARAFON (2 tablets t.i.d. or q.i.d.) in sprains—strains—myositis—whiplash injuries—low back pain

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Precautions: The precautions and contraindications that apply to all steroids should be observed.

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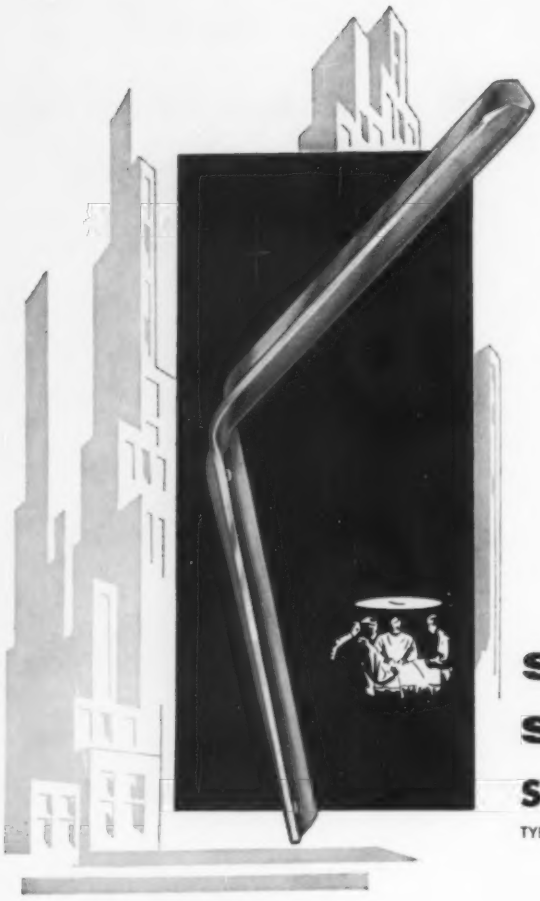

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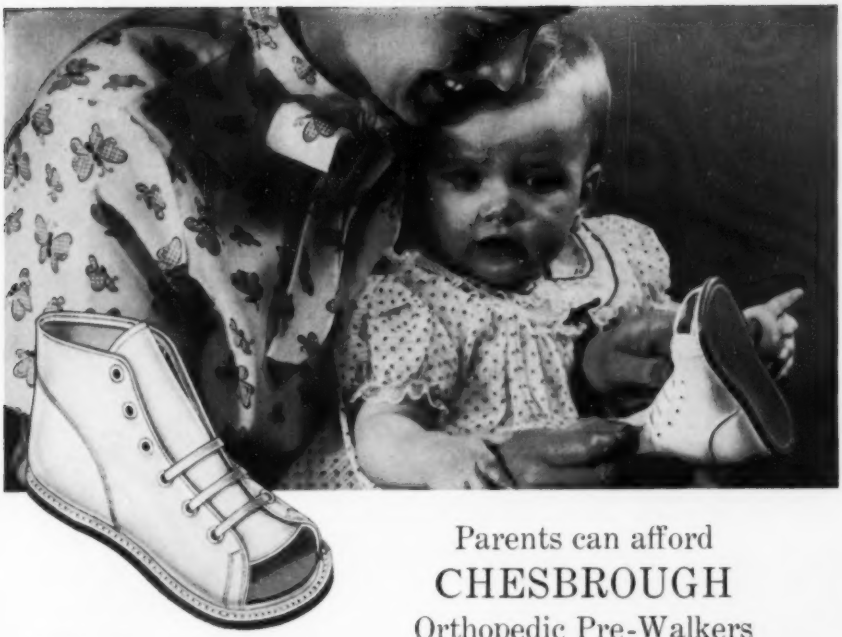

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Orthopedic Pre-Walkers

No. 1400 OPEN TOE
Straight-line symmetrical last, firm heel, no back seam. Adaptable to Denis Browne Splints.

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Lace-to-toe design permits snug, gentle fit. Perfectly smooth inside.

No. 1700 CLUBFOOT, OPEN TOE
Special outflare last, sturdy instep strap to stabilize heel.

Louis C. Weld, President of G. W. Chesbrough Co. . . . "My own personal experience led to the development of the new Chesbrough Shoe."

Here are Orthopedic infants' shoes that meet rigid requirements of corrective footwear yet fit the family budget.

High-quality workmanship and fine leathers with orthopedically correct lasts are combined in Chesbrough Shoes to sell at a moderate price.

Any parent whose child requires orthopedic correction knows the expense is great, as frequent purchase of new shoes is required.

This problem was brought home to Chesbrough's president, Louis C. Weld, three years ago, when a child in his own family needed such a shoe. Recognizing the need for orthopedic shoes at an economical price, Chesbrough Company put their 60 years of shoemaking experience to work and Chesbrough Corrective Pre-Walker Shoes were born.

All shoes in unlined white elk, sizes 000 to 4, narrow and wide. Available in full pairs, split pairs or single shoes (no extra charge for half pairs).

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in the low back syndrome

not only *relieves pain* but also *relaxes taut muscles*

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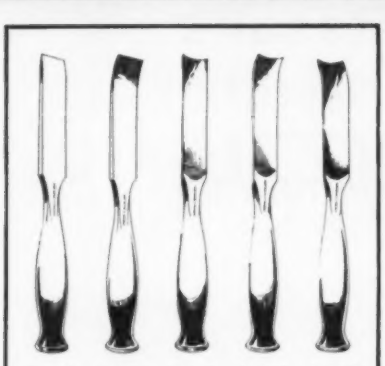

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0-250 SMITH-PETERSEN OSTEOTOME—straight, 8", sizes ¼", ⅜", ½", ⅝", ¾", 1" and 1¼". Stainless Steel.

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A NEW STAINLESS STEEL CASE TO HOLD SMITH-PETERSEN OSTEOTOMES and GOUGES

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CASE 0-295—holds seven Smith-Petersen Osteotomes.

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Cutting edges are protected at all times. Complete unit may be autoclaved.

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WITH CLOSED CIRCULATION AND SUCTION HemoVac®

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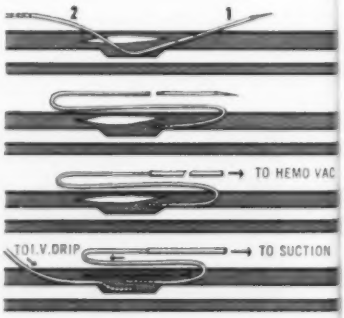

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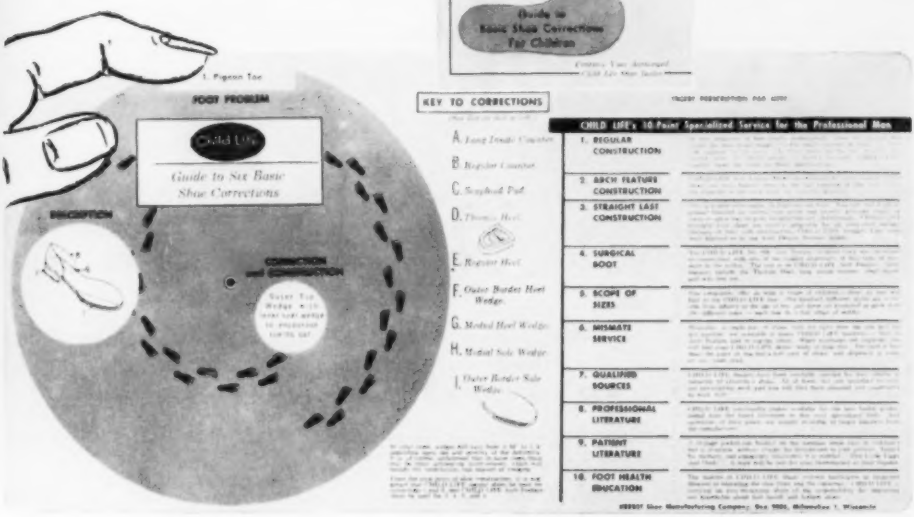
LOOK FOR THE TRADEMARK ⓩ

This is for YOU — doctor
a Guide to Basic Shoe Corrections for Children



The advantages of this new Guide are practical, and they are visual. You see at a glance everything that is involved. Just dial the problem . . . and you automatically receive a diagrammatic description of the correct wedging procedure with a capsule explanation of the correction.

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**Makers of Child Life Shoes for more than 35 years. One of America's great sources of juvenile footwear, both in correction and regular construction.*

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Please send on for my use and that of my office associates __ copies of your Guide to Basic Shoe Corrections for Children.

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Is femoral head replacement advantageous?

Limited originally to salvage procedures, replacement arthroplasty has become increasingly important in the last decade, and is now in many indications regarded as the operation of choice. Many hundreds of case reports clearly define the advantages of hip arthroplasty. [1-12]

"The replacement prosthesis has a very definite place in surgery of the arthritic hip, especially when the head of the femur is essentially gone or inadequate." [9]

In the past 5 or 6 years the well-established criteria for replacement arthroplasty have been notably expanded to include refractory rheumatoid- and osteoarthritides. Femoral head replacements in these conditions offer great promise in those individuals where crippling is severe, or where drug therapy does not seem to facilitate ambulation. Clinical results of numerous hip arthroplasties in rheumatoid- and osteoarthritides have proved highly gratifying both immediately and after lengthy follow-up. [2-8]

Aside from shorter hospital stays, hip arthroplasty offers additional advantages in that secondary surgical procedures are seldom required, nursing care is eased, and the economic burden of the patient is thus reduced. Moreover, prosthetic surgery of the hip affords earlier and more complete ambulation, freedom from pain and limp in most cases, and by providing an almost "normal" hip, enables these patients quickly to return to an active, useful life. [1, 11, 13-20]

Indications:

- Rheumatoid arthritis
- Acute fractures in the aged
- Un-united, long-term fractures
- Delayed non-union
- Acute unstable fracture (Pauwels' 3)
- Osteoarthritis
- Aseptic and avascular necrosis
- Bilateral ankylosis
- Salvage
- Pathologic fractures

REFERENCES: 1. Beegel, P. M.: J. Maine M. A. 49:86, 1958. 2. Moore, A. T.: J. Bone & Joint Surg. 39A:811, 1957. 3. Symposium on Femoral Head Replacement Prostheses, J. Bone & Joint Surg. 38A:407, 1956. 4. Stinchfield, F. E. et al.: J. Bone & Joint Surg. 39A:1043, 1957. 5. Thompson, F. R.: J. Bone & Joint Surg. 36A:489, 1954. 6. Moore, A. T.: South. M. J. 45:1015, 1952. 7. Gaenslen, F. G.: Wisconsin Med. J. 59:181, 1960. 8. Moore, A. T.: In Acad. Orthop. Surg. Instructional Course Lectures, vol. 16, C. V. Mosby, 1959, p. 309. 9. Aufranc, O. E.: Med. Forum, Mod. Med. 27:33, 1959. 10. Thompson, F. R.: In Acad. Orthop. Surg. Instructional Course Lectures, vol. 16, C. V. Mosby, 1959, p. 299. 11. Savastano, A. A., et al.: Rhode Island Med. J. 41:425, 1958. 12. Day, P. L. and Hinchey, J. J.: Clin. Orthop., No. 6, p. 27, 1955. 13. Breck, L. W.: Southwestern Med. 35:207, 1954. 14. Hart, G. M.: J. Lancet 78:369, 1958. 15. Peterson, L. T. and Raad, M. A.: Med. Ann. D. C. 24:233, 1955. 16. Carlander, L. W.: Minnesota Med. 42:566, 1959. 17. Mahoney, J. W. et al.: Am. J. Surg. 95:577, 1958. 18. Barker W. E.: Manitoba Med. Rev. 38:673, 1958. 19. Matchett, F: Rocky Mountain Med. J. 56:47, 1959. 20. Bascom, J. et al.: J.A.M.A. 169:1863, 1959.

Osteoarthritic hip of long standing.

The F. R. Thompson Hip Prothesis In situ.

"Artificial prostheses for replacement of the upper end of the femur deserve a place in our armamentarium of treatment of hip disease...At this stage...the prosthesis has come of age..."[7]

The F. R. Thompson Intramedullary Hip Prosthesis, Catalog No. 6936, is available in head diameters from 1½" - 2¼", in ⅛" increments. For F. R. Thompson instruments, please consult your Austenal catalog, section III, page 7.

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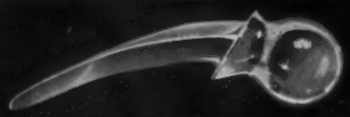
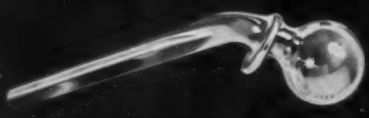
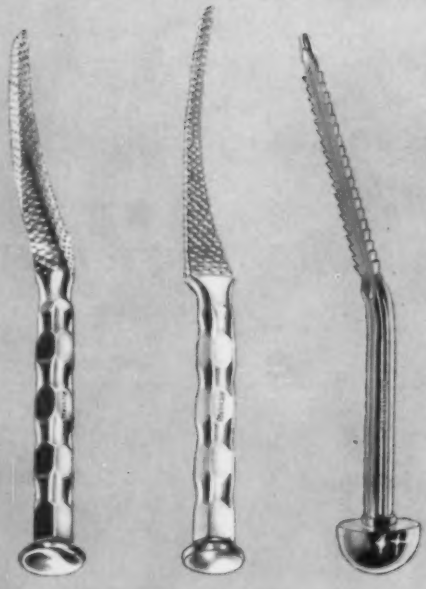


F. R. Thompson Type Hip Prosthesis — Popular choice of surgeons because of its ease of insertion and good results.

Moore Type Hip Prosthesis — Holes in stem allow bone growth through the prosthesis (sometimes filled with bone crumbs before insertion).



Eicher Type Hip Prosthesis — Tri-finned shaft, available in two diameters. Shaft length 5½".



Rasp for F. R. Thompson type prosthesis.

Rasp for Moore type prosthesis.

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Teflon Faced Drivehead — Anodized aluminum body. Totally inert driving face of Teflon. Concave face fits all heads. May be autoclaved.

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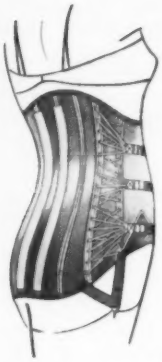

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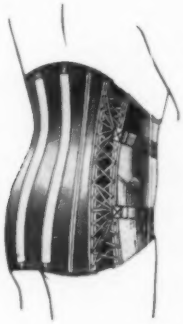

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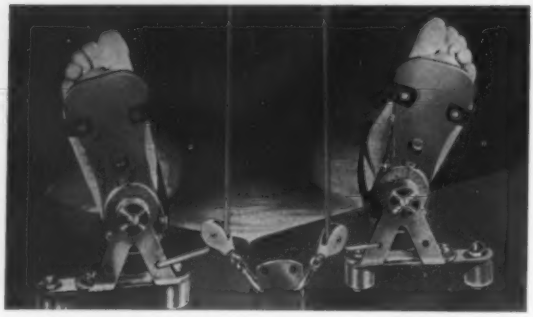

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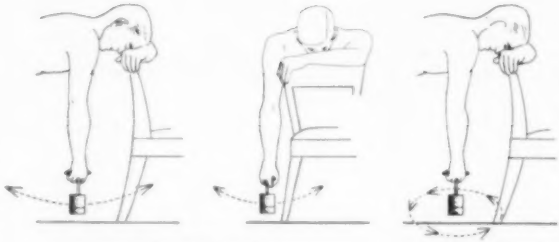

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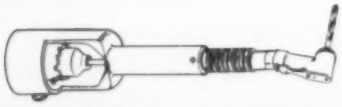

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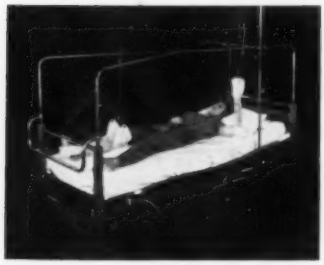

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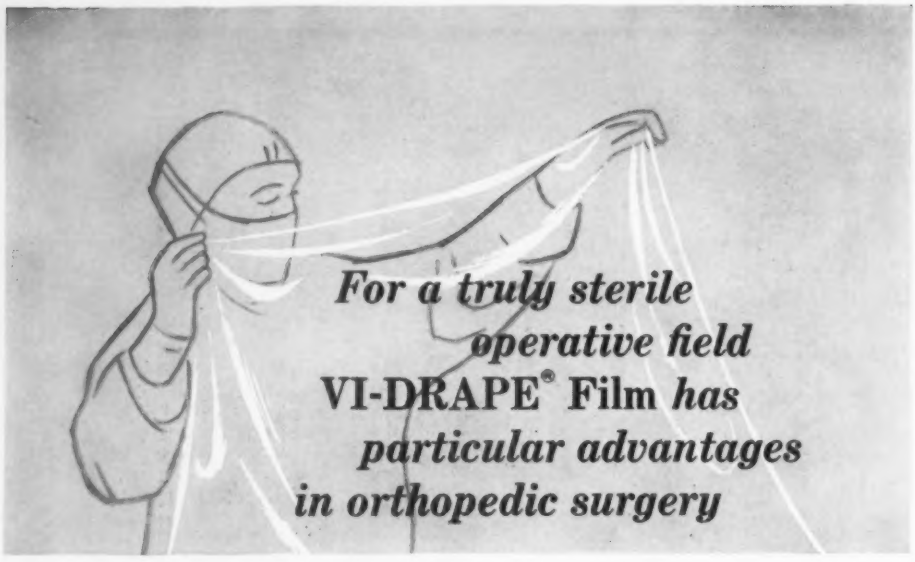

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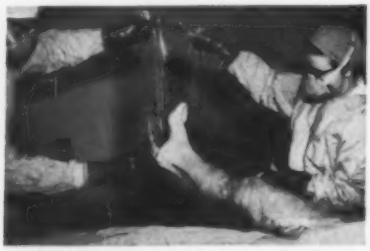

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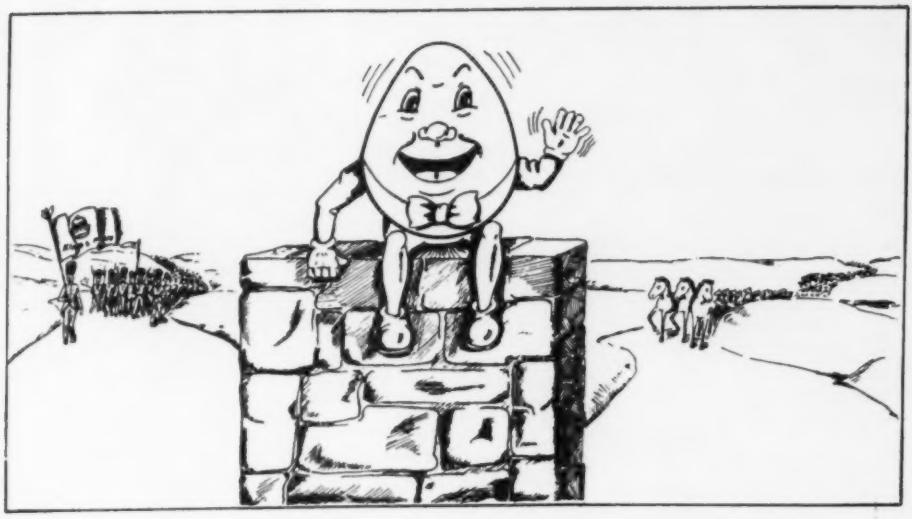

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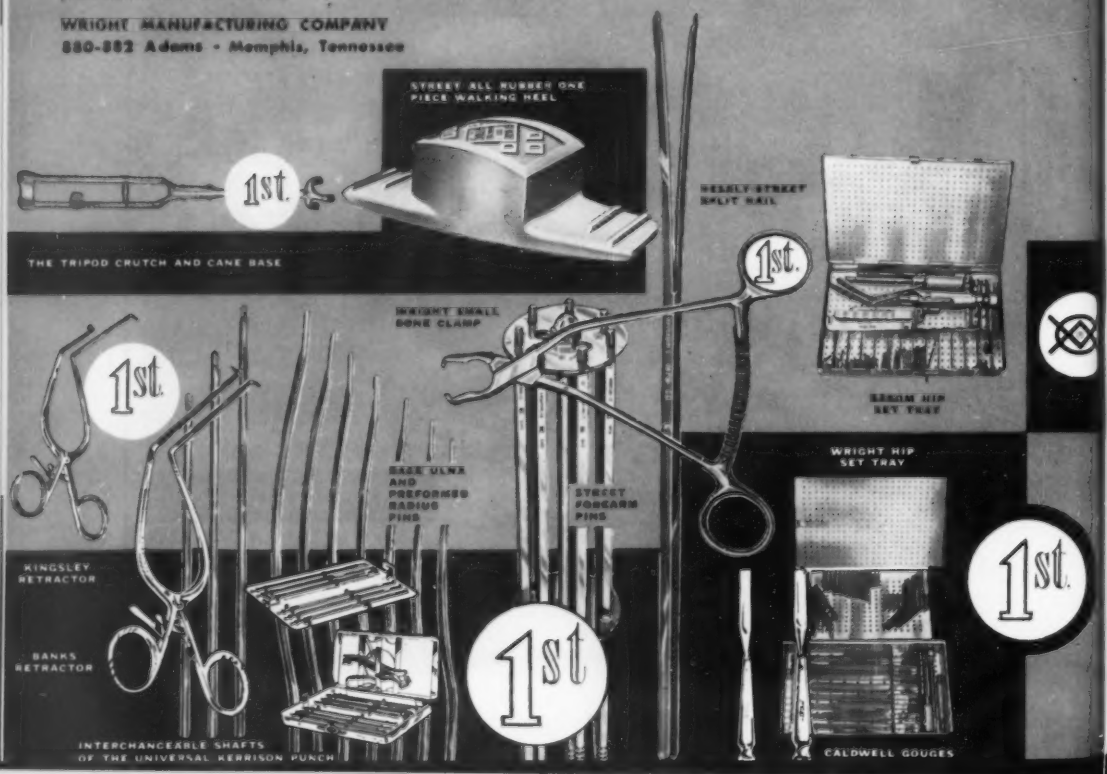

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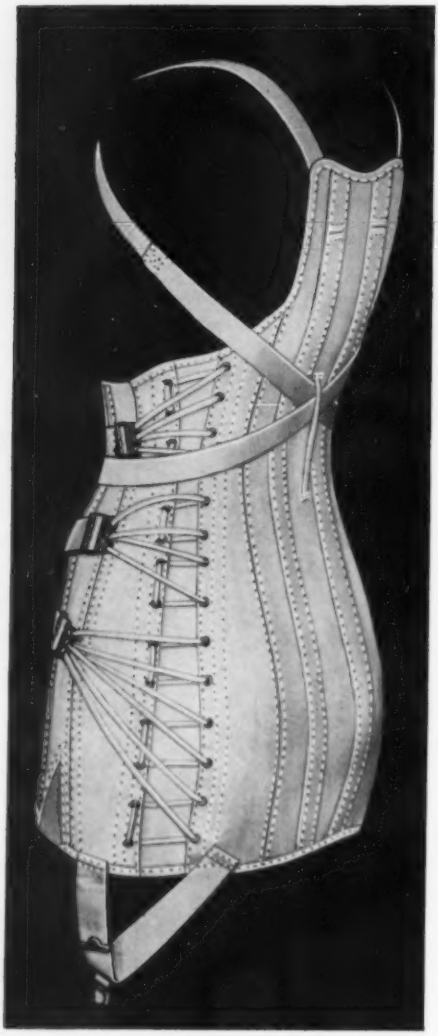

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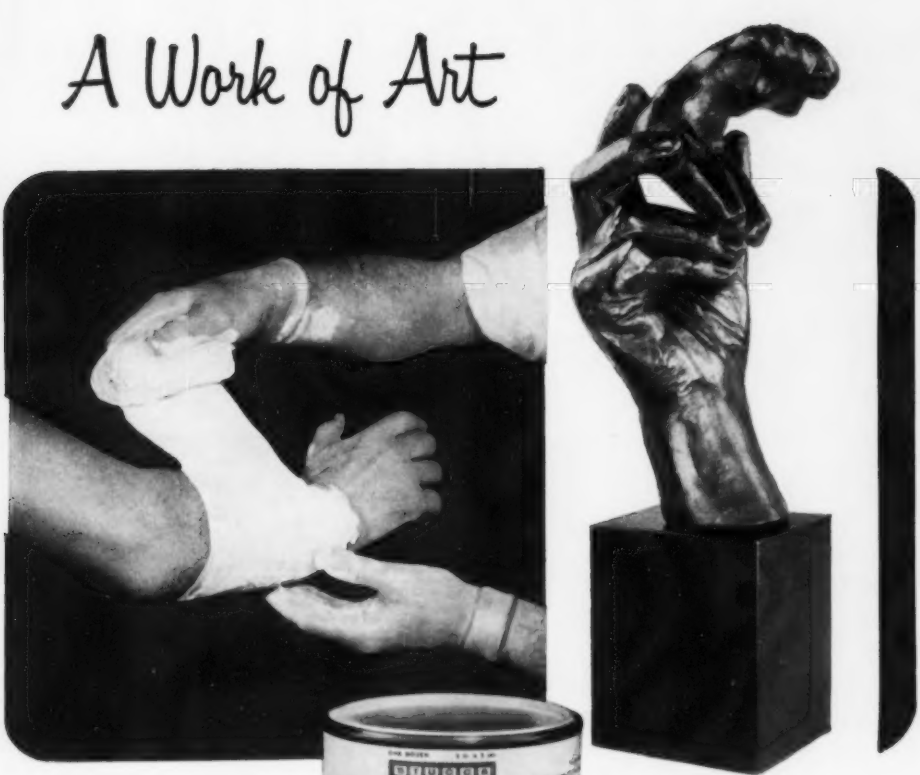

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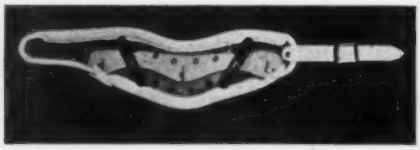

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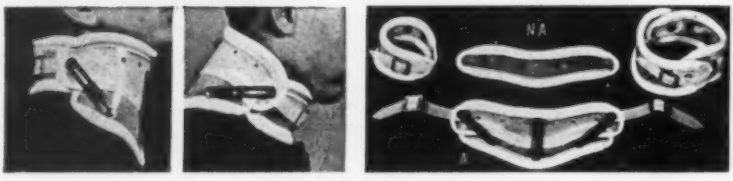

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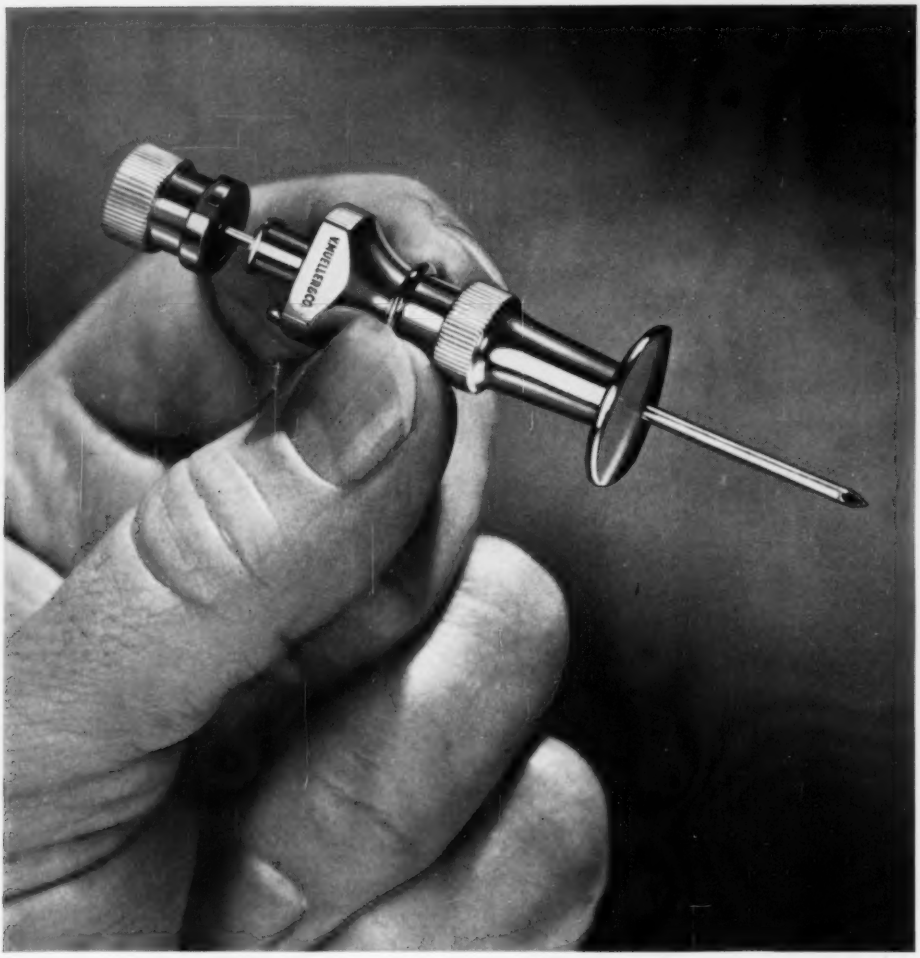

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The Journal of
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Injuries to the Medial Ligaments of the Ankle

Result Study*

BY O. SHERWIN STAPLES, M.D., HANOVER, NEW HAMPSHIRE

From the Mary Hitchcock Memorial Hospital and Hitchcock Foundation, Hanover

For centuries, closed reduction and external splinting have been the traditional treatment for ruptures of the deltoid ligament of the ankle. These injuries are usually caused by lateral dislocation of the talus and ordinarily are accompanied by fracture of the fibula. Recently, several authors [4,6,11,22] recommended open suture of the deltoid ligament as a more satisfactory routine method of treatment for complete tears of this structure, whereas other authors [3,7,12] recommended open surgical treatment for ruptures of this ligament only in special situations. I agree in principle with these latter authors.

In general, at the Hitchcock Hospital, we have treated lateral dislocations of the talus with injury of the deltoid ligament by conservative methods. It has been my impression that major injuries to the medial ligaments of the ankle do well when they are recognized early and treated properly by closed methods. Recent reports recommending routine surgical repair of ruptures of the deltoid ligament stimulated me to review our results in all patients treated for this injury at the Hitchcock Hospital from 1946 through 1958. Only those patients were included who had actual tears of the medial collateral ligament and lateral dislocation of the talus, unequivocally demonstrated by clinical examination or roentgenogram, or by both of these methods of observation. There were 110 such patients.

Over 200 years ago Petit pointed out that medial and lateral dislocations of the ankle almost never are simple injuries. He further pointed out that displacement of the talus cannot occur without fracture of a malleolus or displacement of the fibula, combined with forcible lengthening of the ligaments between the talus and tibia. It is now widely known that ruptures of the deltoid ligament are usually part of a complex of bone and ligamentous injuries, and that in the evaluation of unsatisfactory results it is difficult to determine what element of the complex is responsible. Fortunately, the results in this study were in general quite satisfactory.

The literature on injuries to the ankle was thoroughly reviewed by Bonnin and by Ashhurst and Bromer, and there have been many significant contributions

* Read at the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 1, 1960.

to the subject [14,15,17]. However, it is not the purpose of this study to review the literature or to consider the effects of various forces on dislocation of the ankle. My main interest concerns the ligaments that stabilize the ankle joint on its medial side.

ANATOMY

The ligaments to be discussed include the almost horizontal anterior and posterior tibiotalar ligaments, the more superficial fan-shaped tibiocalcaneal ligament, and the deeper tibiotalar ligament. These ligaments, known collectively as the deltoid ligament, serve to maintain the normal relationship of the talus to the tibia, the various ligaments contributing, more or less, depending on the position of the talus and the nature of the forces applied.

FIG. 1-A FIG. 1-B

Figs. 1-A and 1-B: Stress roentgenograms of anatomical specimens with all ligaments between the tibia and fibula completely severed.
Fig. 1-A: Abduction of foot.
Fig. 1-B: External rotation of foot.

The functions of the deltoid ligament, the interosseous ligaments, and other supporting structures about the ankle were studied very thoroughly by Close in 1956. He found, on the basis of biomechanical studies on fresh anatomical specimens, that unless the deltoid ligament was torn, lateral displacement of the talus in relation to the tibia would not exceed two millimeters, even if all the tibiofibular ligaments were severed. Similar experiments were repeated by us in six fresh specimens after all the soft tissue, except the ankle capsule and ligaments, had been removed. The ligaments were progressively sectioned and the effects of the division of each ligament on the stability of the ankle were observed grossly and by stress roentgenograms. Our findings were similar to those of Close.

Experimental stress roentgenograms showed minimum displacement of the talus, with both abduction of the hind part of the foot and external rotation of the foot in the specimens in which all ligaments between tibia and fibula had been cut, including the distal six inches of the interosseous membrane (Figs. 1-A and 1-B). Similarly, when the anterior capsule, the anterior tibiotalar ligament, and the anterior two-thirds of the tibiocalcaneal and underlying tibiotalar ligaments were also severed, very little displacement was demonstrated on the stress roentgeno-

grams (Figs. 2-A and 2-B). In our anatomical study it was obvious, as it has been to others for many years, that it is impossible to simulate the injuries that occur in the living ankle. However, from these experiments it was obvious that many structures must give way before significant lateral displacement of the talus can result from the usual abducting or externally rotating forces that produce these injuries. In addition to the deltoid ligament, the anterior capsule of the ankle joint, the tibiofibular, interosseous, and transverse ligaments must give way, unless a fracture of the distal part of the fibula releases the holding power of the lateral malleolus.

In the living, by clinical examination, one can find various degrees of ankle instability as the result of injury—from the ankle which under anesthesia has only a slight amount of increased lateral mobility to the completely unstable

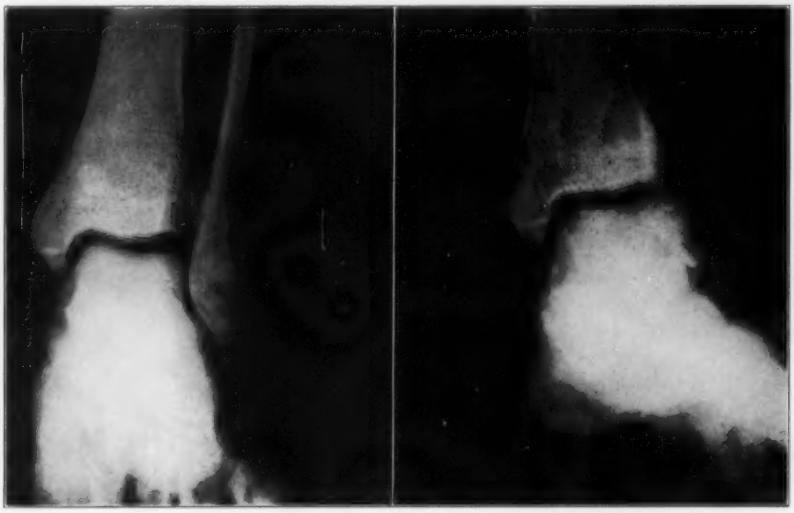

FIG. 2-A FIG. 2-B

Figs. 2-A and 2-B: Stress roentgenograms of an anatomical specimen with all the ligaments between the tibia and fibula, the anterior joint capsule, the anterior tibiotalar, and the anterior two-thirds of the tibiocalcaneal and underlying tibiotalar ligaments severed.
Fig. 2-A: Abduction of foot.
Fig. 2-B: External rotation of foot.

ankle. Studies of anatomical specimens aimed to correlate the types of ligament injury with the various degrees of ankle instability seen in the living after injury did not produce any valid information.

Clinically, the frankly unstable ankle and the ankle with only slightly increased lateral motion are easily recognized, but I found no good way to identify ankles with intermediate degrees of instability. For the purposes of this study I have classified as *complete* ruptures of the deltoid ligament those ankles with one centimeter or more of so-called clear space between the medial malleolus and talus on anteroposterior roentgenograms made with the leg held in 20 degrees of internal rotation—the so-called mortise view. This division is perhaps too arbitrary and is certainly subject to challenge, but it separates the more severe from the less severe injuries. Clinical examination and stress roentgenograms were also used to estimate the severity of these injuries. Fifty-seven injuries of the deltoid ligament were considered to be complete; fifty-three, incomplete. Interestingly enough, as shown by the results in this study, this division seemed to be of no significance

FIG. 3-A
Clinical test: lateral ankle stability.

FIG. 3-B
Clinical test: anteroposterior ankle stability.

FIG. 4-A
Patient D.W. Original roentgenogram, made elsewhere.

since the poorer results were equally divided between the complete and incomplete groups.

DIAGNOSIS

Diagnosis is no problem when fixed deformity exists, but frequently no displacement is obvious. The original roentgenograms so often show only a so-called simple oblique fracture of the fibula. A tear of the deltoid ligament, known to us as the invisible injury, can easily be missed. In this series of 110 patients, this diagnosis was missed initially nineteen times; eight of these errors in diagnosis were made in patients seen first in our own clinic. Clinical examination, which is so often neglected when a fracture can be demonstrated by roentgenogram, is of prime importance. Immediately after injury one can usually demonstrate abnor-

FIG. 4-B

FIG. 4-C

Fig. 4-B: Original stress eversion roentgenogram (made elsewhere).
Fig. 4-C: Roentgenogram (made by us) after one week in a boot cast. Note lateral subluxation of the talus, not apparent in the original roentgenograms.

FIG. 4-D

Final reduction.

mal mobility of the talus in relation to the tibia, but pain and spasm soon prevent such testing. However, when spasm is present, a tear of the deltoid ligament and an unstable ankle must be ruled out when tenderness and swelling are present on the medial and anterior aspects of the ankle, with or without fracture of the fibula.

It is generally agreed that roentgenograms should include anteroposterior, lateral, and oblique views with the leg in internal rotation to show the ankle mortise. These examinations may show some degree of dislocation, which indi-

FIG. 5

Stress roentgenogram to test the integrity of the deltoid ligament.

FIG. 6

A method of maintaining full inversion after reduction, in preparation for application of the cast. The thigh and leg are horizontal and the hip is in external rotation. The patient is tipped toward the affected side.

cates the true diagnosis, but if dislocation is not apparent and there is any doubt concerning the stability of the joint, examination of the ankle or stress roentgenograms under anesthesia are necessary to test the integrity of the ligaments.

The technique of testing ankle stability should include the determination of the total range of ankle and foot motion on the two sides with a comparison of the degree of lateral and rotatory movement of the talus in relation to the tibia (Fig. 3-A). This maneuver tests the retaining function of both medial and lateral ligaments of the ankle.

In addition, anteroposterior play of the ankle should be determined in order to test the competence of the anterior ankle capsule and both collateral ligaments, particularly their anterior elements [1] (Fig. 3-B).

In stress roentgenography, we have routinely used only one position to test for abnormal lateral excursion of the talus. This procedure was tested on a cadaver and seems to be quite trustworthy. With the patient face up, the leg is internally rotated 20 degrees at the hip, and the ankle is placed in 10 degrees of

plantar flexion. The examiner, with his hands protected by lead gloves, then forcibly externally rotates and abducts the foot and a roentgenogram is made (Fig. 5). Both ankles should be tested in this fashion. Other stress roentgenograms to test the anterior joint capsule and lateral ligaments can be obtained as indicated.

The importance of stress roentgenography is well established, but the performance of the test is the clinician's responsibility. Poorly made stress roentgenograms can be misleading. As an illustration: One patient came to the hospital with routine roentgenograms of the ankle that showed a low fracture of the fibula

FIG. 7

After reduction of a lateral fracture-dislocation of the ankle. The cast is in full inversion.

with no dislocation of the talus. One stress roentgenogram with the foot held in valgus position also showed no dislocation, but no anesthesia had been used during the test. The deltoid ligament was assumed to be intact and a boot cast was applied. Lateral dislocation of the talus was obvious in a roentgenogram made through the cast one week later (Figs. 4-A through 4-D).

TREATMENT

Since rupture of the deltoid ligament is almost always a part of a complex of injuries to bone and ligament, all elements of the injury require treatment. The usual form of treatment for this complex of injuries at the Hitchcock Hospital during the past thirteen years has been closed reduction and fixation in a plaster cast. An occasional dislocation cannot be reduced because of an interposed rolled-up deltoid ligament or displaced posterior tibial tendon [8], but as a rule, complete reduction of the dislocation is quite easy to achieve, although sometimes hard to maintain. Open operative treatment on bone or ligament was used in fresh injuries only when closed manipulation failed or when there were special problems, such

FIG. 8-A

Patient P.B., after reduction of a lateral fracture-dislocation of the left ankle. There is anterior subluxation.

FIG. 8-B

After second manipulation and cast fixation in dorsiflexion and internal rotation of the foot. The subluxation is corrected.

as open fractures. With an external rotation injury resulting in dislocation of the talus on the tibia, I have found that slight adduction of the foot with molding of the cast is less trustworthy for the maintenance of reduction than a position of full internal rotation and adduction of the foot. The position of inversion goes back, certainly, as far as Dupuytren [9]. Figure 6 shows the position presently employed at the Hitchcock Hospital for holding the foot and leg when applying a cast. Full internal rotation can be maintained by holding the toes while the weight of the leg in the side-lying position provides counter traction. This position facilitates application of the cast.

Ordinarily, a toe-to-groin cast is used. The ankle shown in Figure 7, held in full internal rotation and varus position, had had previous reduction and a cast applied in a less severe position, but this had failed to maintain the reduction. In the position shown, complete reduction of the lateral dislocation of the talus caused by rupture of the deltoid ligament and the oblique fracture of the fibula

FIG. 8-C

One year later there is calcification of the deltoid ligament and the interosseous ligaments and proliferative change of the anterior lip of the tibia.

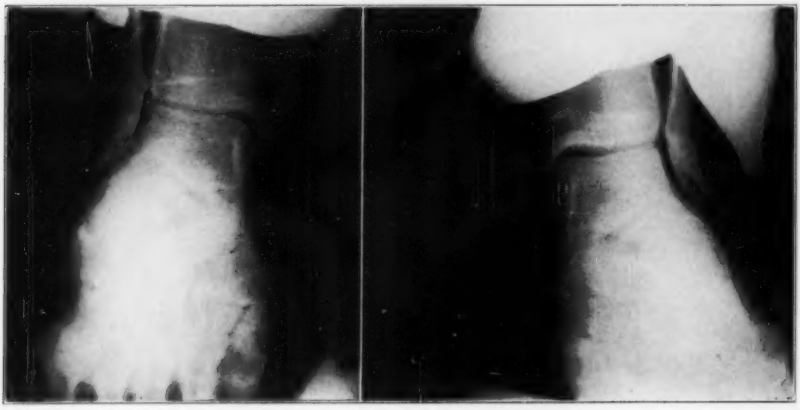

FIG. 8-D

Stress eversion roentgenograms, made at the same time as Fig. 8-C, show less talar tilt on the injured side (left). The clinical result was good; the patient had no disability.

was achieved. However, if this extreme position is maintained too long it may cause lasting disability. As a rule, a new cast in less inversion is applied in three to five weeks.

The ordinary treatment after reduction of these injuries at the Hitchcock Hospital includes bedrest for from two to seven days depending on symptoms, a long cast worn from three to five weeks, then a cast to the knee with the foot in slight inversion for six to eight weeks more. A walking cast is not ordinarily used. After cast removal, an exercise program to restore flexibility and strength is most important—for weeks or even months. Weight-bearing with or without crutches is allowed according to the patient's functional capacity. The total duration of immobilization in plaster in this series averaged ten weeks.

As in many problems, there can be no standard method of therapy. Occasionally, when the injury has caused, primarily, a valgus stress on the heel with little

FIG. 9-A

Patient P.B.B. Original roentgenogram shows complete fracture-dislocation of the posterior lip of the tibia and high fracture of the fibula.

external rotation, the ankle can be immobilized more effectively if the heel is placed in full varus position and the foot is held in only slight internal rotation. Examination under anesthesia or a postreduction roentgenogram will make this clear.

During the thirteen years covered by this study, ninety-three patients with ruptures of the deltoid ligament were treated by closed reduction. Of these, seventy-seven were treated with internal rotation of the foot and sixteen with full varus position of the foot and slight internal rotation. In nine of these ninety-three patients, a second manipulation was required after roentgenograms of the ankle in the plaster cast showed an unacceptable position (Figs. 8-A through 8-D).

RESULTS

Clinical Material

The patients in this series of ruptures of the medial ligaments of the ankle were relatively young, and men were the predominant group. Thirty-five were Dartmouth undergraduates; eighteen were students from other colleges who

FIG. 9-B

At follow-up, seven years after injury. The patient now manages a farm; he has no symptoms and no disability.

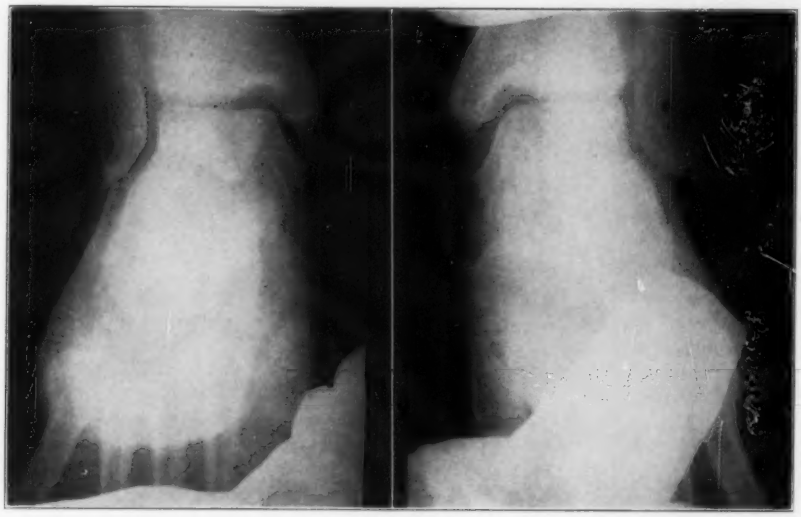

FIG. 9-C

Stress eversion roentgenograms made seven years after injury show no instability of the talus.

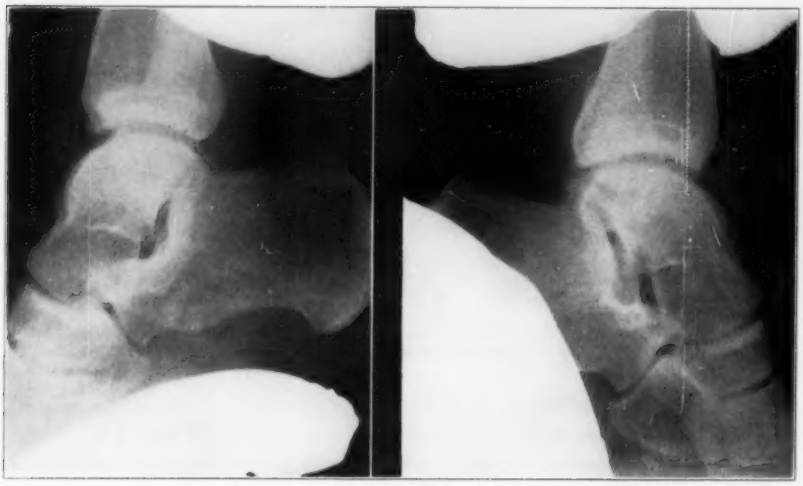

FIG. 9-D

Stress roentgenograms made seven years after injury to test anteroposterior stability shows it to be normal.

were injured while skiing in the vicinity of Hanover. Only twenty-eight patients did heavy labor. Sixty-six patients were under thirty years of age and only eight were over sixty. The average age at time of injury was 31.2 years. Skiing was the most common cause of injury (Table I). The type of injury encountered is shown in part in Table II. In the 100 patients in whom rupture of the deltoid ligament was accompanied by fracture, the low, short oblique fracture of the fibula that is associated ordinarily with the external rotation injury, was most common. A variety of other fractures were seen, including the high fibular fracture of Maisonneuve (Figs. 9-A through 9-D).

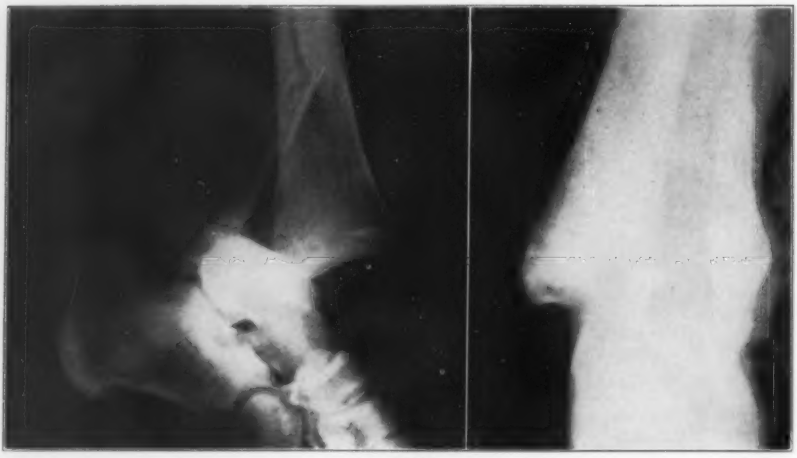

FIG. 10-A

Patient J.C. Original roentgenograms showing fracture-dislocation of the left ankle.

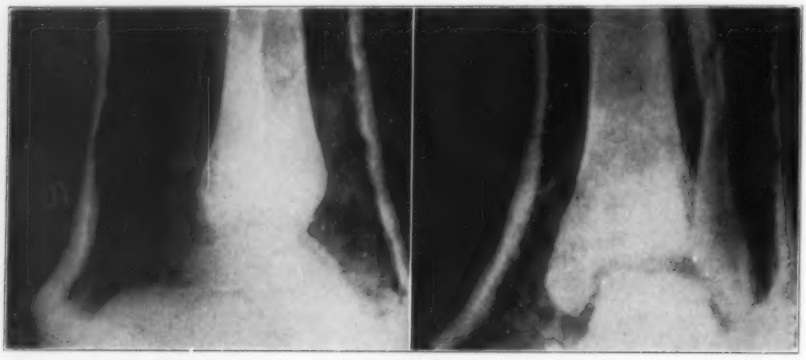

FIG. 10-B

Roentgenograms made after reduction. Position: full internal rotation and abduction. Note the talar tilt in the anteroposterior view, indicating a tear of the lateral ligaments of the ankle.

A lateral or posterolateral dislocation of the ankle is often associated with a fracture of the posterior lip of the tibia and sometimes with a fracture of the lateral margin of the distal end of the tibia, as demonstrated by Cooper over a century ago from autopsy material. With fractures involving the lateral margin of the tibia, the tibial fragment is displaced laterally with the fibula, leaving the tibiofibular ligaments intact. Only ten of the 110 patients with rupture of the deltoid ligament in this series did not have some type of fracture.

Most of the ankles in this series showed clinical and roentgenographic evidence of injury to capsular and ligamentous structures, in addition to the deltoid ligament. In seventy-three there were ruptures of the tibiofibular ligaments; in sixty-three, rupture of the anterior capsule of the ankle; in five, ruptures of the lateral ligaments; and in twelve, complete dislocation of the talus, all with fracture of the fibula. In twenty-two, the nature of the ligamentous and capsular injuries, in addition to rupture of the medial collateral ligament, was unknown. Ankles with both the medial and the lateral collateral ligaments torn, as shown by roentgenographic evidence of both medial and lateral instability, present un-

FIG. 10-C

Roentgenogram made when cast was changed, ten days after injury, showing reduction of inversion and adduction of the foot.

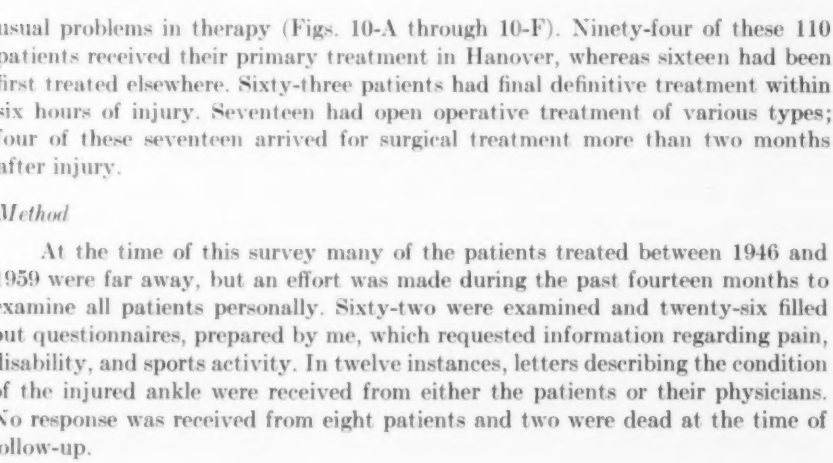

FIG. 10-D

Roentgenograms made at follow-up, seven years after injury. There are no symptoms and no disability in the left ankle.

usual problems in therapy (Figs. 10-A through 10-F). Ninety-four of these 110 patients received their primary treatment in Hanover, whereas sixteen had been first treated elsewhere. Sixty-three patients had final definitive treatment within six hours of injury. Seventeen had open operative treatment of various types; four of these seventeen arrived for surgical treatment more than two months after injury.

Method

At the time of this survey many of the patients treated between 1946 and 1959 were far away, but an effort was made during the past fourteen months to examine all patients personally. Sixty-two were examined and twenty-six filled out questionnaires, prepared by me, which requested information regarding pain, disability, and sports activity. In twelve instances, letters describing the condition of the injured ankle were received from either the patients or their physicians. No response was received from eight patients and two were dead at the time of follow-up.

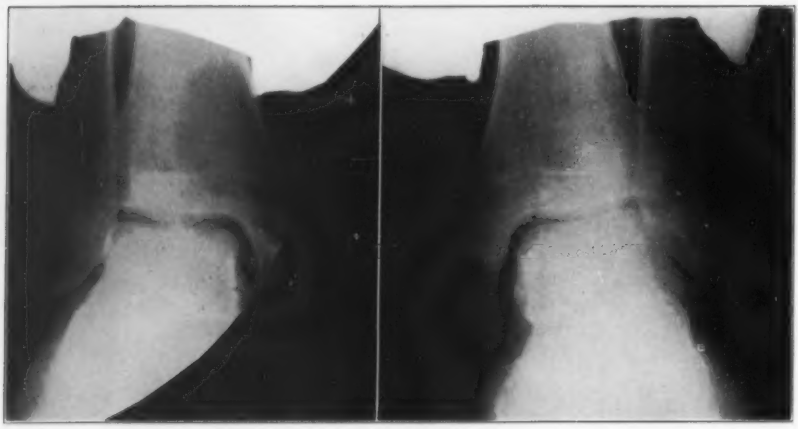

FIG. 10-E

Stress eversion roentgenograms made at follow-up show both ankles to be normal.

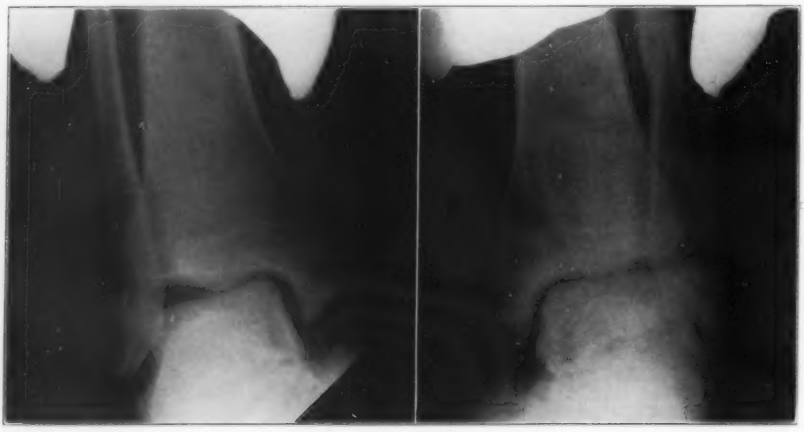

FIG. 10-F

Stress inversion roentgenograms made at follow-up. The left ankle is stable but in the right ankle there is instability due to a recent untreated ligament injury. This resulted in recurrent sprains and mild disability.

Follow-up examinations of sixty-two of the 110 patients were done by me. Each patient was questioned concerning the time required for recovery after injury, his present symptoms (stability, pain, strength, or swelling), further injury, participation in sports, or any disability related to his occupation or to sports.

The physical examination took into consideration gait, posture of the foot, deformity, swelling, tenderness, comparative strength of the patient's two ankles, as judged by the examiner, range of motion, and an evaluation of the lateral and anteroposterior stability of the ankle.

Routine anteroposterior, lateral, and oblique roentgenograms were made of sixty-one patients. Additional bilateral stress roentgenograms with the foot held in external rotation and abduction were made of fifty-eight patients. The stress roentgenograms were made under my supervision.

Data from the work sheets of the 100 patients followed were transferred to statistical punch cards, collected, and processed by a sorting machine. For pur-

TABLE I

TYPE OF ACTIVITY AT TIME OF INJURY

Activity	No. of Patients
Skiing	58
Misstep and fall outdoors	22
Automobile accident	8
Fall indoors	5
Football	5
Other vigorous sports	4
Fall from height	4
Miscellaneous	4

TABLE II

ASSOCIATED FRACTURE AT TIME OF LIGAMENT RUPTURE

Type of Fracture	No. of Fractures
Low, oblique fracture of the fibula, beginning at the joint line	63
Low, oblique fracture of the fibula, above the joint line, diastasis	19
High fracture of the fibula, diastasis	10
Low transverse fracture of the fibula	7
Posterior fracture of the lip of the tibia	8
Fracture of the lateral articular margin of the tibia	1

TABLE III

OPERATIVE TREATMENT: SEVENTEEN PATIENTS

Treatment	No. of Patients
Bone Surgery	
Early Surgical Treatment	
Open reduction of the posterior lip of the tibia	3
Open reduction of the fibula	4
Late Surgical Treatment	
Bone graft for non-union of the fibula	2
Excision of osteophyte of the fibula	1
Excision of osteophyte of the tibia	1
	11
Ligament Surgery	
Early repair of the deltoid ligament	3
Late repair (over two months) of the deltoid ligament	3
	6

TABLE IV

CLOSED TREATMENT: CHANGES SENE ON FOLLOW-UP ROENTGENOGRAMS

Calcification, margins of tibiofibular ligament	40
Calcification, deltoid ligament	26
Bone proliferation, tip of medial malleolus	26
Degenerative changes in ankle, mild	15
Spur formation, anterior lip of tibia	21
Spur formation, posterior lip of tibia	11

poses of statistical analysis the seventeen patients treated surgically were excluded. The remaining eighty-three patients treated by closed reduction were studied statistically as a group, with particular attention to the various factors

FIG. 11-A

Original roentgenograms of Patient I.P., who had an open wound on the medial side of the left ankle. The deltoid ligament was so severely damaged and grossly contaminated that it was necessary to excise it during débridement. The dislocation and fractures were reduced by manipulation and held in inversion by a toe-to-groin cast.

FIG. 11-B

Follow-up roentgenograms, one year after injury.

in injury, treatment, and follow-up findings that might influence the result. The data were reviewed by Professor Thomas Kurtz of the Mathematics Department of Dartmouth College; none of the factors which might have influenced the results was found to be statistically significant. This is perhaps to be expected in this small group of variably complex injuries with a high proportion of good results. The most significant finding was that only eleven patients had any disability.

Symptoms and Disability

The group of patients treated by operation was too small and too diverse to be analyzed (Table III). The average time to follow-up examination was 5.5 years, with a range from eleven months to twelve years. In all, there were seventeen patients operated on because of ruptures of the deltoid ligament and associated injuries. Surgical treatment was confined to the bone structures in eleven patients, and in six only the deltoid ligament was repaired. The results in the group treated by bone surgery were all excellent, with the exception of two patients

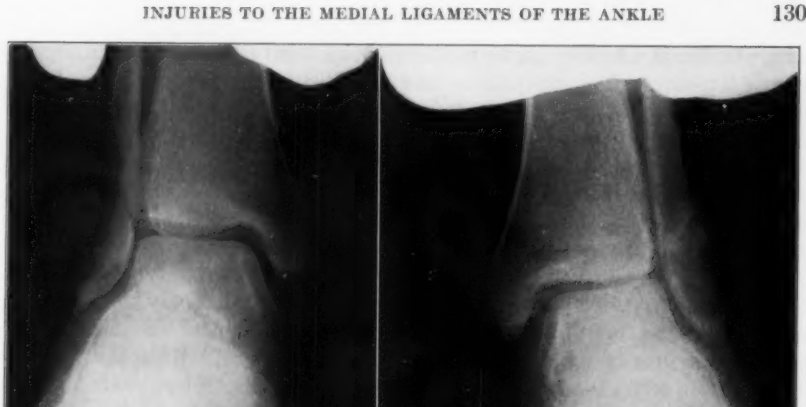

FIG. 11-C

Stress eversion roentgenograms at follow-up, one year after injury. There is a slight (two millimeters) increase in the width of the space between the talus and the medial malleolus of the left ankle.

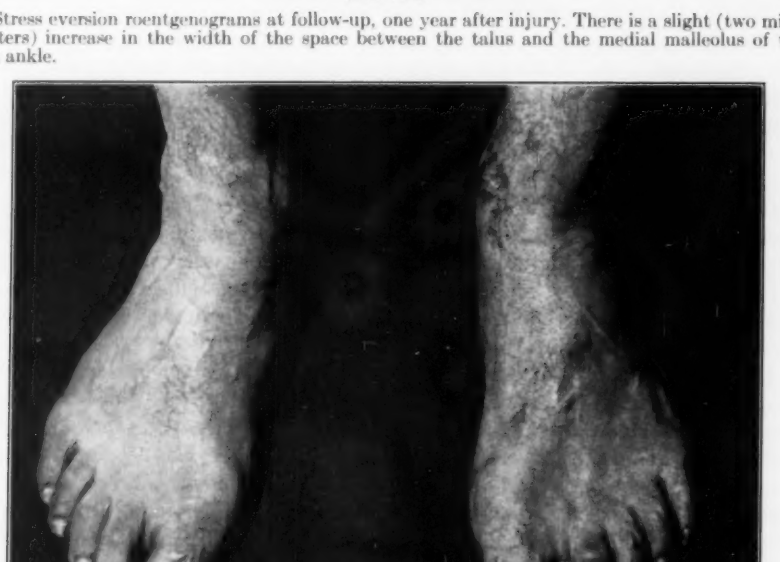

FIG. 11-D

Photograph made at follow-up, one year after injury. There is no deformity in the left ankle, which is stable clinically. There is, however, weakness on prolonged activity. Pain is absent, and the ankle is still improving.

who had slight disability when engaging in sports and two others with slight, non-disabling pain.

Of the six patients who had open operations for repair of the deltoid ligament, one who had had a fresh open dislocation of the ankle had mild disability at the time of follow-up. Two others, one operated on late and one early, had non-disabling pain. The patient who was treated by early suture of the deltoid ligament had pain on hard running over rough terrain, but was not disabled; in fact, he

was a member of a college ski team. He was examined one year after injury and was still improving. The other three patients of this group were symptom-free.

Of more interest are the remaining eighty-three patients who were treated conservatively. The average time to follow-up was 5.9 years, with a range from one year to thirteen years. In this group, the time required for recovery could not be determined for the patients surveyed by mail. It is of interest to note that in the forty-seven others, full recovery took place in only twenty-two during the first six months after injury. Several were still improving at the end of one year.

In the eighty-three patients treated by closed reduction only eleven (13 per cent) had any disability, and this was mild. All of these patients had pain, but only one had pain in the region of the deltoid ligament. In three the pain was lateral, and in seven it was general in the ankle. Eleven other patients had non-disabling mild pain related to unusual activity, and six others had mild aching in wet weather, again without disability. Three additional patients reported that the affected ankle was not quite so trustworthy as the uninvolved ankle, but they had no pain or disability.

Peroneal and tibial muscle weakness was found in four patients who had disabling pain and in two of the three patients who had mild instability of the ankle without disability. This finding was not judged significant in this study; but in a group of ankle sprains studied previously [5], weakness of these muscles seemed to have some relationship to the persistence of symptoms.

The findings in regard to participation in sports showed that fifty-five patients (66 per cent) were able to engage in vigorous sports such as skiing, football, baseball, and tennis. To our knowledge, only two patients could not participate in sports at all. No patient in this group had any disability for walking, and none had changed his occupation or noted any occupational disability. Eighteen patients did not participate in sports through choice. There was no information in regard to sports activity from the remaining eight patients.

None of the eleven patients who were rated as having slight disability was handicapped as far as occupation was concerned. There were four housewives who were all fully active, although two were rather elderly. Of the seven remaining patients one was a lawyer, one a librarian, one a teacher, one a student, one a store manager, one a railroad-yard worker, one an Air Force officer, and one a submarine commander. Although not handicapped for their work, five of these eleven patients consistently had pain in the ankle when walking on rough ground and avoided doing this; four had pain after long walks; one had pain only when running and hence avoided this; and one stated that he could not fully trust his ankle although he could play tennis and water ski. There were no clinical or roentgenographic findings that could be correlated with these symptoms and disabilities.

Follow-up Examination

In forty-seven of the eighty-three patients treated by closed means and available for clinical examination there were no findings that could be related significantly to the results. Slightly increased pronation of the foot was found in three patients and in one, mild deformity of the fibula was obvious. Mild thickening of the deltoid ligament was present in thirty patients, and mild tenderness was found over the anterior, medial, or lateral aspects of the ankle in eight.

In this group of forty-seven patients, fifteen had slightly less than normal strength in their peroneal and tibial muscles and six of these fifteen patients with weakness had pain, instability, or some disability in their ankles. Only

fourteen ankles had full motion but of those with loss of motion only four had lost more than 10 degrees in any direction. Pain on forced motion was found in nine patients, but this was not severe and two of these nine patients did not complain of pain on use. The loss of motion in this group was comparable to that found in patients who had had open operations. Increased lateral mobility of the ankle was found in ten patients but four of these had no complaints of pain or disability. No ankle showed anteroposterior instability. All of the abnormalities found were minor; none was statistically significant or even suggestive as a cause of disability, except for the muscular weakness already mentioned.

Follow-up Roentgenograms

Obvious changes due to fracture were noted on follow-up roentgenograms. In addition, there were many changes related to ligamentous injury (Table IV). These changes were not statistically significant when checked against results, and were similar to the changes seen on the follow-up roentgenograms of the ankles treated by open reduction.

The distance of the medial articular surface of the talus from the medial malleolus was measured on oblique roentgenograms. A two-millimeter lateral shift of the talus was found in five patients, and in one there was a three-millimeter lateral shift. Of these six patients, three had disability or pain which was suggestive, although perhaps not significant.

DISCUSSION

Although no statistically valid conclusions can be drawn concerning the best method of treating a rupture of the deltoid ligament of the ankle, this study has shown that conservative management can be effective. With no disability for work or ordinary walking in any patient and only mild disability in eleven of the eighty-three patients treated conservatively who were available for evaluation, it seems reasonable to expect a good result from non-operative treatment.

Surgical treatment of the deltoid ligament is clearly indicated if closed reduction does not replace the talus to its proper position. However, in the usual complex injury in which rupture of the deltoid ligament occurs, suture of the ligament alone is only partial treatment. Of the ten patients who had ruptures of the deltoid ligament without fracture, only five had injuries which were apparently limited to the deltoid ligament alone; two had tears of the anterior capsule of the ankle joint and three had tears of the tibiofibular ligaments, in addition to the ruptures of the deltoid ligament. Solitary injury to the deltoid ligament is, therefore, relatively uncommon. In the usual injury which involves the triad of rupture of the deltoid ligament, fracture of the distal end of the fibula, and rupture of the anterior capsule, along with partial tearing of the tibiofibular ligament complex, all the injured structures must be treated. The treatment of this complex of injuries involves either closed manipulation and external splinting alone or, if necessary, a combination of open and closed reduction and splinting. Open surgical treatment of all elements of this injury, including deltoid-ligament suture, control of tibiofibular diastasis by a screw, as Vasli and others recommended, and internal fixation of all fractures seems unduly complicated under ordinary circumstances.

Although the advisability of suture of acute avulsion injuries of the knee is generally accepted [20], the routine operative repair of a torn deltoid ligament is anatomically, and I think practically, a different matter because of the possibility of maintaining the ankle in an overcorrected position and because of the usual combination of bone and ligament trauma. In my experience, meticulous replacement

of the deltoid ligament does not appear to be essential. In one of the patients in our series, who was operated on because of a badly contaminated open wound, the deltoid ligament was completely excised during débridement of the wound. One year later, this patient had a stable ankle; he could walk three miles while hunting and was working regularly in a mill, on his feet all day long. He had weakness and instability after prolonged exertion, but was still improving. Obviously, the scar tissue which must have replaced the deltoid ligament has functioned adequately, at least so far (Figs. 11-A, 11-B, 11-C, and 11-D).

Some of the findings in the patients treated by closed methods are not desirable—for example, the lateral shift (several millimeters) of the talus in six patients. It is possible that in these ankles there may develop degenerative changes and increasing disability at a later date [10,12,13,23,24]. Whether more careful conservative treatment or open repair of the deltoid ligament and other structures would have given better results in these six patients is a matter for speculation.

Our limited experience with open surgical repair of injuries of the deltoid ligament indicates that good results can be obtained by this method. However, it is misleading to compare these results with those of patients treated late, and poorly, by closed methods. If ruptures of the deltoid ligament are recognized early and treated well by conservative measures, one can doubt that significantly better results can be achieved by routine open surgery.

SUMMARY

A study of injuries of the deltoid ligament of the ankle in anatomical and clinical material indicates that solitary injury to this ligament is rare; it is more commonly found along with other injuries, including a fracture of the distal part of the fibula. A survey was made of patients treated at the Hitchcock Hospital for major injuries of the deltoid ligament (110 patients in a thirteen-year period). Almost all of these patients were treated by closed reduction and external splinting. In eighty-three patients treated conservatively and with follow-up data available, only eleven had any disability and this was mild. A small number treated by open surgery showed comparable results. On the basis of this study it would seem that the majority of ankles with a complex of injuries including rupture of the deltoid ligament can be treated well by closed reduction and external splinting.

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DISCUSSION

DR. CHARLES J. FRANKEL, CHARLOTTESVILLE, VIRGINIA: Dr. Staples demonstrates clearly that the intelligent coordination of expert hands and sound judgment can still bring about good results, and that the knife, the hammer, the nail, and the screw may be dispensed with in a majority of cases.

I agree with Dr. Staples that the lost art of conservative treatment should be revived and that the end results that may be expected from such intelligent treatment are for the most part good to excellent. Our series of 270 cases (140 in amateur athletes) bears out Dr. Staples' statements completely.

I would question only some minor inconsistencies. First, Dr. Staples stated that he was unable to apply clinically the information that he obtained through anatomical study—a dividing line between complete and incomplete ligament ruptures. If that is so, and I agree with Dr. Staples that it is so, then why bother to make stress roentgenograms prior to treatment? Why subject the patient to anesthesia? Particularly since the results obtained in ankles with a wide spread, as compared with a lesser spread, of the mortise were not too different. We doubt that it is possible to get adequate stress roentgenograms without anesthesia. Secondly, Dr. Staples has arbitrarily used toe-to-groin casts. I would be interested in knowing why. It has been our experience that we have been able to maintain position just as well in a well fitted leg cast, particularly in the elderly patient. We haven't been troubled by the supposed rotation or torsion that takes place within a leg cast. Thirdly, Dr. Staples states that surgical treatment of the deltoid ligament is indicated if closed reduction does not replace the talus to its proper position. Yet, he admits that a complex situation exists in the ankle when the deltoid ligament is ruptured. Many other ligaments, as well as the capsule, are usually torn and surgical treatment of the deltoid ligament alone is not adequate and repair of the total soft-tissue injury is usually impractical. How much spread of the mortise will Dr. Staples accept before he attempts surgical intervention? It has been our experience that a spread of a few millimeters does not of necessity imply a painful or a disabling disability.

DR. OTTO E. AUFRANC, BOSTON, MASSACHUSETTS: The occasional partial failure of tight healing in ligamentous injuries has led us to consider and at times to carry out surgical sutures of these structures. In the surgical repair of ruptures of the deltoid ligament all is not neat and easily resutured. It is often no more than a realignment of the strands and approximation of torn ends with some quilting sutures. Occasionally, there is a nice avulsion from bone which cleanly reapproximates by suture and we feel rewarded by our astuteness and surgical skill. Strangely humbling, however, is the fact that we may find the results of either suturing procedure to be the same.

The results of healing on a basis of complete or incomplete tears or ruptures would thus seem to need some other factor, for example, how the tears occurred and how much is torn. We feel our reward more when we can remove the torn end of a ligament from the joint—even if we wonder why it might not have been possible to manipulate the ligament free without surgery. In fresh injuries this can and probably does happen.

Stress roentgenograms on anatomical specimens for any joint injury are useful and always

(*Continued on page 1316*)

Arthrodesis of the Ankle with Particular Reference to the Gallie Procedure

A Review of Fifty Cases

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The Gallie ankle fusion was precisely described by the late W. E. Gallie in 1948. His paper was devoted entirely to surgical technique and recorded the evolution of his procedure in twenty-five cases over an eight-year period. He made no attempt to present a follow-up evaluation. For some peculiar reason, this publication has been ignored continually in the literature, and recent articles

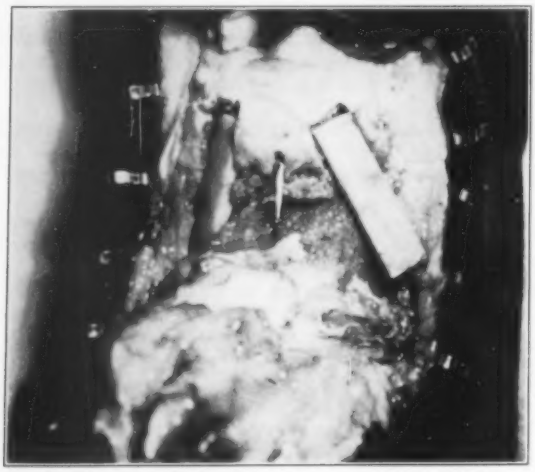

FIG. 1-A

FIG. 1-B

Principles behind the Gallie fusion as demonstrated on cadaver. Dorsal structures have been removed. Medial graft is being driven into position.

dealing with ankle fusion have failed to include it in their references. Orthopaedic surgeons often confuse this operation with the original Gallie subtalar fusion in which he employed a posterior approach. The Gallie ankle fusion is carried out through dual anterior incisions with the anterior grafts countersunk into the medial and lateral sides of the ankle-joint mortise (Figs. 1-A and 1-B).

Surgical arthrodesis of the ankle, although not an uncommon procedure, is certainly not a routine daily orthopaedic operation in my community. However, over the past ten years, I have encountered, either primarily or in follow-up, fifty patients who have undergone this procedure. Twenty of the fifty fusions were performed by me and in identical fashion, employing a modification of the original Gallie technique. The remaining thirty fusions were carried out in this hospital by three recognized orthopaedic surgeons, each of whom limited his technique to one of three other methods to be described (Table I).

A review of the literature consistently brings out two points. First, many

authorities, while making worthwhile contributions, have labored under the impression that, prior to the publication of their own particular articles, the problem of securing surgical arthrodesis of the ankle was almost insurmountable.

Second, it is always dangerous to claim originality when describing any operative technique [9]. A wave of irritation followed the simple description by Watson-Jones of his anterior arthrodesis, with many other authors laying claim to originality. A similar outburst accompanied the excellent article by J. Crawford Adams on transfibular ankle fusion (1948), with other authors supplying ample evidence that this approach had been clearly discussed by Horwitz in 1942, who, in turn, gave credit to Goldthwait's work in 1908.

We have, therefore, been cautious in giving Dr. Gallie credit for originality in his procedure. Barr and Record and Mead employed the dual anterior incisions, but similarity ceased therewith. Mead carried out osteotomy of both malleoli, whereas Barr and Record employed a simple anterior fusion.

TABLE I

TECHNIQUES IN FIFTY FUSIONS

Anterior	Gallie	20
Anterior	Watson-Jones	14
Anterior	Compression	3
Lateral	Crawford Adams	13

Review of the pathological lesions demanding surgical arthrodesis of the ankle indicates that trauma is predominant. Trauma was the basic etiological factor in twenty-nine of the fifty fusions included in this study. Fusion for poliomyelitis was confined to the ankle joint alone in nine cases, fusion was done for tuberculosis and insidious osteo-arthritis in four and five cases, respectively, and fusion was done for miscellaneous reasons in three cases.

FUSION BY OTHER METHODS

Anterior

Since this paper concerns primarily the Gallie fusion, I shall only touch briefly on other techniques. The anterior Watson-Jones procedure with certain reasonable modifications was carried out in fourteen of the fifty cases. The entire procedure is perhaps more difficult than it seems at first glance. Both its originator and I have discarded it in favor of less demanding techniques.

There were three technical difficulties that were experienced in the fourteen anterior fusions done by the Watson-Jones procedure: (1) poor fitting of the graft in the talus; (2) danger of distraction of articular surfaces by the graft; and (3) creation of a defect and consequent weak point in the lower end of the tibia in an effort to secure a sufficiently large bone graft.

These difficulties are exemplified in one patient who was left with a large defect in the tibia (Fig. 2), and in a second patient who suffered a fracture through the donor site nine years after arthrodesis. Creation of a bone defect in close proximity to an ankylosed joint invites such complication unnecessarily.

Despite the technical difficulties of this procedure, only one of the fourteen patients had a failure of fusion. However, the period in plaster after this procedure was much longer than that after other methods; in several cases the period of immobilization was six or seven months, compared with the average period of three months in plaster after both the transfibular and the Gallie procedures.

The anterior compression technique of Charnley yielded indifferent results

in three ankles (one delayed fusion, one failure of fusion, and one fusion in unsatisfactory position). There were too few cases to permit assessment of this method. These indifferent results were possibly caused by the reluctance of the surgeon to boldly divide tendons, nerves, and blood vessels across the front of the ankle joint. This resulting inadequate exposure may have concealed lack of exact bone contact between the components of the ankle joint. Without such contact, the value of compression is lost. Charnley recently reported on fifty-nine such fusions. Only in isolated areas in Canada has compression arthrodesis of the ankle by Charnley's method gained popularity.

Lateral

Thirteen patients underwent transfibular arthrodesis. This appears to be an excellent operation, being reasonably simple in technique and affording a good

FIG. 2

FIG. 3

Fig. 2: Precarious defect at donor site following anterior arthrodesis.
Fig. 3: Transfibular fusion with reinforcing medial graft. The inaccessible medial aspect of the joint still remains partially open.

view of the ankle joint. One failure four months after operation stemmed from inadequate consolidation over the medial side of the joint. This region is relatively inaccessible from the transfibular approach. Because of this, the method was modified in the final seven patients. A small incision was made over the medial side, a drill hole was created traversing the medial joint space, and a small tibial graft was inserted between the medial malleolus and talus. The need for this reinforcing graft is open to question, although several pioneers in the use of this procedure now employ this reinforcing graft (Fig. 3).

The transfibular arthrodesis has been a worthwhile addition to ankle-fusion techniques. It may be considered the procedure of choice in many instances but is unsuitable in the following: (1) in the growing child where a fibular graft must not cross the tibial epiphyseal plate; (2) in the person whose skin over the lateral aspect of the fibula is in a precarious condition from previous trauma or disease; and (3) in the person whose residual pathological condition is concentrated along the medial side and thus it is relatively inaccessible from the lateral approach.

With reference to technique, care must be taken not to produce distraction

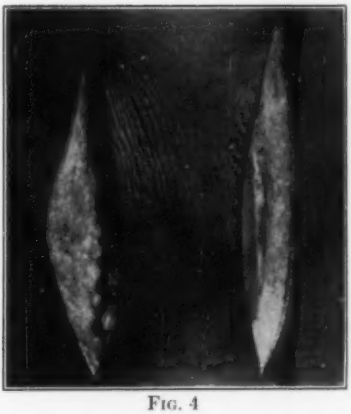

FIG. 4

FIG. 5

Fig. 4: Dual anterior incisions. These appear more lengthy than standard incisions due to plantar-flexed position of ankle.

Fig. 5: Excellent visualization of the ankle joint with gentle retraction of intervening skin flap.

FIG. 6

FIG. 7

Fig. 6: Accurately placed graft stopping short of lower tibial epiphyseal plate.

Fig. 7: Lateral view of ten-year follow-up. Absorption of grafts is noted as well as absence of reaction about the staple. An excellent clinical result.

between the talus and tibia when the fibular graft is placed and to secure proper coaptation between the fibula and the other bones of the ankle joint. Division of the fibula must be clean, since a noteworthy finding in four patients at the follow-up examination was tenderness over the resected end of the remaining proximal part of the fibula.

Of the thirteen patients with transfibular fusions, eleven were classified as having excellent or good results. These eleven patients had no pain or minimum pain, had an intact fusion in the planned position, and walked with no limp or, at most, only a slight limp when wearing shoes. One patient who had pain of an annoying nature in the lateral compartment despite a solid fusion was classified as having a fair result. The remaining patient was classified as a failure because fusion failed to occur along the medial side of the joint, and a second attempt at

arthrodesis was made. This patient still had pain three years after the second procedure.

GALLIE ANKLE FUSION

The basic Gallie technique was adhered to in my twenty cases but there were several modifications that will be discussed later. I might add that the official blessings of Dr. Gallie were constantly with me during this work, and it was a great disappointment to me that he did not live to comment on this paper.

The ankle joint is conveniently exposed by a short vertical incision over each malleolus. One incision is just medial to the anterior tibial tendon, and the second incision is just lateral to the peroneus tertius tendon (Fig. 4). The joint capsule is exposed, and the joint is opened on each side (Fig. 5). The articular cartilage of both surfaces is removed through the dual incisions. A mortise is then prepared above the medial aspect of the ankle joint, approximately three-eighths of an inch in width and one and one-half inches in length, running well into the lower end of the tibia and directed slightly lateralward at its proximal end. If the epiphysis is still open, the mortise must stop short of the lower tibial epiphyseal plate, which may readily be identified in the operative field (Fig. 6). A similar mortise is then prepared in the lateral side, preserving, if possible, the upper fibers of the inferior tibiofibular ligament to prevent lateralward drifting of the fibula.

At this stage, the joint is placed in the desired position for fusion, and a single staple is driven across the joint midway between the beds that have been fashioned for the grafts. This staple anchors the talus to the tibia. I have found the addition of a staple a useful adjunct in the Gallie technique. It secures the ankle in an optimum and planned position for fusion, and, in addition, prevents the talus from displacing in a lateral or medial direction when the cortical struts are being wedged into position. Two staples are used if the bone is unduly osteoporotic. To date, refrigerated bone has been used in all my cases, and I make no apologies for its use. The abundant cancellous environment provides ideal circumstances for revascularization. The entire operative procedure is simplified by use of preserved bone, and the lower end of the tibia is not weakened by removal of bone. No additional bone is employed.

The postoperative management of the patients was quite conservative; an above-the-knee plaster cast was used for six weeks and then was followed by a below-the-knee cast for an additional six weeks. Wound healing was excellent, and in no case was skin viability in the intervening tissue between vertical incisions ever in question.

The present operation differs in three respects from Dr. Gallie's original technique: (1) a staple is used; (2) refrigerated bone is employed; and (3) adjacent cartilage is removed.

I am quite certain that Dr. Gallie would have disapproved of all three changes. However, I have found the staple of great value in controlling the talus while the tibial grafts are being driven home, and refrigerated bone has behaved in an exceedingly satisfactory fashion. The third change—the removal of articular cartilage—is the most debatable and perhaps complicates the procedure more than necessary. There is no question that once thorough removal of articular cartilage has occurred, difficulties may arise in fitting the talus to the lower surface of the tibia. Dr. Gallie showed this removal to be unnecessary, since the articular cartilage covering the weight-bearing surfaces gradually will thin out and collapse, allowing the growth of bone from the tibia to the talus. However, in many cases, slight degrees of varus or valgus deformity must be corrected. This entails removal of articular cartilage in moderate amounts. In other in-

FIG. 8

FIG. 9

Fig. 8: Fusion with persistent severe internal rotation deformity. Subsequent external rotation osteotomy was necessary.

Fig. 9: Accurate placement of grafts and staple with lower tibial epiphyseal plate not traversed and remaining open.

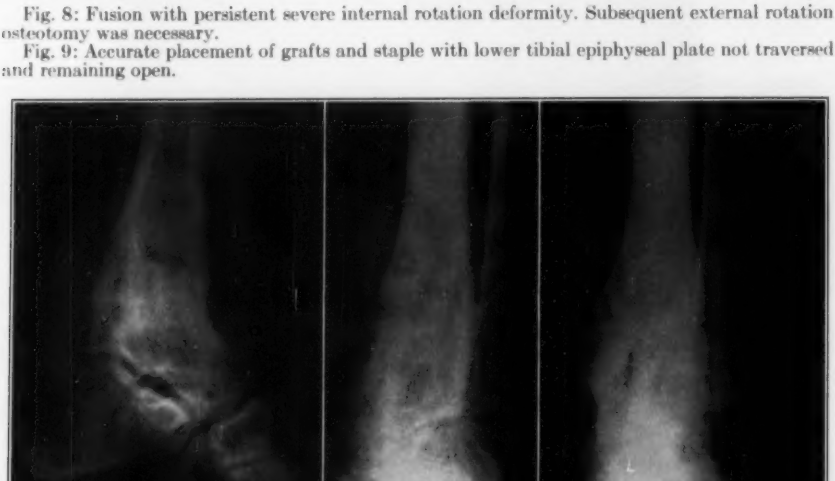

FIG. 10

A seventeen-year follow-up of second fusion performed by Dr. Gallie. Although articular cartilage was never removed, solid arthrodesis has occurred across the joint space.

stances, the excellent exposure on both sides of the ankle provided by the Gallie approach made me unable to resist removal of such cartilage.

In a follow-up of our twenty patients with Gallie procedures, all patients were seen during the three months prior to writing this article. Seventeen patients had very satisfactory results, with a minimum of constant complaints. Two patients had indifferent results, with pain in both the lateral compartment of the ankle and in the mid-tarsal region. These two patients undoubtedly had previous mid-tarsal arthritis that was aggravated by ankle fusion. One ankle examined after ten years showed excellent fusion with absorption of the grafts (Fig. 7). One patient had a poor result due to my inability to correct an internal rotation deformity by ankle fusion alone (Fig. 8). This man subsequently had a rotation

osteotomy of the tibia, but continues to have pain and swelling about his ankle. I feel reasonably certain that no method of ankle fusion alone could have corrected this deformity at the ankle-joint level. Seven patients in this group underwent fusion of the ankle prior to closure of the distal tibial epiphyseal plate (Fig. 9). Three of these patients have been followed until the completion of growth and four have been followed for from one to three years since fusion. Although accurate roentgenographic measurements of growth since fusion have not been obtained, all epiphyseal plates remained open after the procedure, and there was no gross evidence of interference with growth.

FIG. 11

A common area for postoperative pain following ankle fusion.

The Gallie ankle fusion may be employed in such patients in whom an anterior fusion, a compression arthrodesis, or a transfibular fusion is contraindicated because of damage to the lower tibial epiphyseal plate. With care, grafts can be countersunk into the epiphysis, and a staple can be employed without damage to the plate. In retrospect, it now appears that a staple perhaps should not have been used in this group, since it is most difficult to secure sufficient grasp on the epiphysis without crossing the epiphyseal plate.

Inquiry might be made as to when an ankle fusion alone is indicated in a foot afflicted with poliomyelitis. In adults with a paralyzed foot in neutral position, the ligaments may be sufficiently tight that only fusion of the ankle joint is needed. In children, if a foot-drop deformity is present without any varus or valgus deformity and with no muscle available for transfer, ankle fusion alone may be sufficient, if it is remembered that the older the child is at the onset of poliomyelitis, the less stretch there is to tendons and ligaments, and the better candidate he is for ankle fusion.

GENERAL OBSERVATIONS

It was somewhat disappointing that no remarkable general observations were forthcoming from this follow-up of fifty ankle fusions. Although the majority of fusions were ultimately satisfactory, a variety of complaints were commonly associated with this procedure before the end result was obtained. These included annoying swelling, localized areas of tenderness, rigidity of the foot, and difficulty in initial adjustment to shoes. The average final result was not obtained until eight to nine months after the removal of plaster.

Gait

The great majority of patients with successful fusions walked well; in fact, so well that, when wearing shoes, little or no limp was noted. Minor compensatory knee bend could be noticed on careful scrutiny, and, conversely, if there was weakness of the knee, compensatory recurvatum deformity was observed, particularly when the ankle had been fused in slight excess of equinus position. In compliance with a standard pattern, an attempt was made to fuse female patients in a position of 5 to 7 degrees more equinus deformity, whereas male patients were fused at a right angle. It would seem, however, that there is no rational method for establishing the optimum angle for fusion. In several female patients the ankle was fused in only three to five degrees of equinus deformity, yet these patients walked very well in moderately high heels. Other women varied the height of the heels they wore with no major discomfort or change in gait pattern.

Compensatory Movement

Rigidity of the ankle inevitably changes the gait pattern. Ankle fusion, undoubtedly, creates excessive strain on neighboring joints about the foot, notably the subtalar and mid-tarsal joints. Excessive lipping or spur formation was commonly noted at the talonavicular joint in follow-up roentgenograms, regardless of what technique had been used. One must not routinely attribute this complication to the arthrodesis, however, since early follow-up roentgenograms in several instances revealed this condition to be already present at the time of the fusion.

Regardless of the type of fusion, the most natural gait was secured in those patients in whom extreme compensatory movement at the mid-tarsal and anterior tarsal joints developed.

During this follow-up study I was most fortunate to come across the second Gallie fusion performed by Dr. Gallie himself. Seventeen years after operation the outlines of the grafts were still visible. Extreme compensatory movement had developed in the mid-tarsal joint; this was associated with slight fragmentation of the navicular bone (Fig. 10).

Exclusive of technical errors at operation, the poorest results occurred in patients in whom arthrodesis of the ankle was inflicted on a foot already burdened with a pathological condition in neighboring joints. Too often this resulted in a semirigid foot, incapable of compensatory movement, and, hence, in subtalar pain.

Postfusion Pain

The pain after ankle fusion is puzzling and noteworthy; it has usually been localized, lateral-compartment pain, just anterior and medial to the lateral malleolus. One-third of the patients in follow-up complained of pain in this region that ranged from being minor and inconstant to steady (Fig. 11). The easiest explanation is to attribute such pain to the subtalar joint where a pathological condition coexists. However, in recent follow-up studies of fractured calcanei with subtalar fusions there was also localization of pain to this area in a high percentage of cases [3]. It would seem that pain in the foot is inherently reflected to this region after trauma or arthrodesis, although there is no anatomical explanation of this phenomenon.

CONCLUSION

Ankle fusion may be accomplished in a satisfactory fashion by a number of standard techniques.

There is still a place for the choice of technique, depending on the situation.

The Gallie ankle fusion with or without modifications is a useful addition and yielded a minimum of complications in twenty patients.

An analysis of fifty ankle fusions revealed sufficient postoperative complaints to emphasize the need for careful patient selection.

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DISCUSSION

Injuries to the Medial Ligaments of the Ankle

(Continued from page 1307)

instructive. Dr. Staples has pointed out the limitations of such studies and takes into account these factors in his analysis. Some of the specific limitations should be mentioned.

The living body with circulating fluid and warmth has an inherent elasticity that slowly disappears when circulation stops and death, rigor mortis, and drying take over to approximate the rigidity of a mummy. So it would follow that cutting ligaments and other structures would show looseness of varying degree, depending on both the time after death and the warmth of the specimen and in the number of things cut. The more things cut, torn, or broken about a joint, the looser it will be. Such a statement needs no support.

In Dr. Staples' clinical cases he gives me a fine chance to quibble and quibbling it is. The invisible injury can be explained on rotation of the talus in the mortise. No one would quarrel with the decision to change the cast; all would applaud the new position of the mortise and none (I hope) would suggest suturing this hidden injury.

The diagnosis is often easy. At other times we assume there are varying degrees of ruptures of the ligament in various places. The question arises: Should we test the remaining ligament to the point of probably increased and possible total destruction? In this point I suggest gravity stress testing with gentle assistance in the anesthetized patient, as a principle in any stress testing. With this suggestion, I simply want to agree slightly with the value of gentle stress testing.

I agree with Dr. Staples that full recovery from this injury takes a long time. Only twenty-two patients recovered before six months and several were still improving after one year. After severe ankle injury, there seems to be a good chance to lose some motion which is not missed by the patient. This allows us a margin for good and excellent results for which we are grateful.

I wish to congratulate Dr. Staples on his very neat and concise study and I agree entirely with his conservative attitude.

Roentgen Aspects of Injuries to the Pisiform Bone and Pisotriquetral Joint

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Recently, our attention was drawn to a succession of injuries involving the pisiform bone and parapisiform area. A perusal of the literature revealed only the briefest consideration of injuries to this area. Our material includes thirteen fractures of the pisiform bone and a number of abnormalities of the pisotriquetral-joint space manifested by widening, asymmetry, or both of these changes, which

FIG. 1

Semisupine view of the wrist of a normal boy, twelve years old. Note the peculiar ossification pattern of the pisiform bone. (Similar findings were present in the opposite pisiform bone.)

were considered to indicate subluxation of this joint. However, there are no well defined criteria for the roentgenographic diagnosis of subluxation of this joint, and, in order to formulate these criteria, an accurate roentgenographic delineation of the kinematics of this joint is necessary.

It is the purpose of this communication to report our findings in the thirteen fractures of the pisiform bone and other abnormalities found in the pisotriquetral

FIG. 2-A

FIG. 2-B

Fig. 2-A through 2-J: Positioning.
Figs. 2-A and 2-B: Dorsipalmar projection.

FIG. 2-C

FIG. 2-D

Lateral projection.

joint in association with fractures and other injuries of the wrist. In addition, a study of the normal kinematics of the pisotriquetral joint is reported, and roentgenographic criteria for the diagnosis of subluxation of this joint are proposed.

ANATOMICAL FEATURES

The pisiform bone, considered by many as a sesamoid [7], is somewhat conical in shape and lies on the medial end of the volar aspect of the proximal row of carpal bones. The volar surface of the pisiform bone is round and roughened and furnishes attachment for the transverse carpal ligament, the tendon of the flexor carpi ulnaris, and the origin of the abductor digiti quinti muscle. The flexor carpi ulnaris tendon extends beyond the pisiform bone as two fibrous bands forming the pisohamate and the pisometacarpal ligaments. Dorsally, the pisiform bone articulates solely with the triquetral bone. The distal portion of the volar surface of the triquetrum articulates with the entire dorsal surface of the pisiform [6].

The pisotriquetral joint is an arthrodial diarthrosis and is enclosed in a small, but usually loose and tough capsule, the fibers of which fuse on the medial side with one fascicle of the ulnar collateral ligament and on the lateral side with the median extension of the transverse carpal ligament [15].

In human beings, the mean age and standard deviation for the appearance

FIG. 2-E

FIG. 2-F

Dorsipalmar position with ulnar deviation and a slight (10-degree) tilt of the ulna upward.

FIG. 2-G

FIG. 2-H

Palmar-dorsal semisupine position: 10 to 30 degrees from the lateral, with the ulnar side down, and the thumb up and out.

FIG. 2-I

FIG. 2-J

Tunnel view.

of the center of ossification of the pisiform bone is eight years and nine months, plus or minus one year and three months [19]. Normally, the bone is fully developed by the time a person is twelve years of age. Ossification of the pisiform bone may originate from multiple centers and give rise to peculiar bone patterns [14] in this age group (Fig. 1).

TABLE I

DATA ON PISIFORM FRACTURES

Case No.	Type of Fracture	Isolated or Associated with Other Fractures of Wrist	Articular-Surface Involvement	Pisotriquetral-Joint Space	Type of Injury
1.	Linear	Isolated	Yes	Normal	Direct trauma
2.	Comminuted	Isolated	Yes	Widened, loss of parallelism (subluxation)	Direct trauma
3.	Linear	Triquetral fracture	Yes	Normal	Direct trauma
4.	Impacted and comminuted	Isolated	Yes	Normal	Direct trauma
5.	Simple chip (distal end)	Isolated	No	Normal	?
6.	Linear	Isolated	Yes	Normal	Direct trauma
7.	Linear	Isolated	Yes	Not visualized	Muscle pull
8.	Comminuted	Fracture of distal part of the radius (Colles')	Yes	Normal	Direct trauma
9.	Comminuted	Triquetrum and hamate fractures	Yes	Normal	Direct crush injury
10.	Comminuted and impacted	Isolated	Yes	Normal	Direct trauma, muscle pull, or both
11.	Simple chip (distal end)	Impacted fracture of the distal end of the radius (Colles')	Not visualized	Not visualized	Direct
12.	Simple chip (distal end)	Fracture of base of first metacarpal and radius; perilunar subluxation	Yes	Normal	Direct crush, muscle pull, or both
13.	Impacted and comminuted	Fracture of the distal end of the radius (Colles')	Yes	Normal	Direct

ROENTGENOGRAPHY OF THE PISIFORM BONE

Roentgenographically demonstrable injuries of the pisiform area are easily overlooked on casual inspection of routine roentgenograms and are often missed because of poor technical detail, improper positioning, or an inadequate number of projections. In the roentgenographic study of the wrist, the following views are suggested: dorsipalmar, with the wrist in neutral position (Figs. 2-A and 2-B); lateral (Figs. 2-C and 2-D); dorsipalmar with ulnar deviation of the wrist and slight elevation of the ulna produced by 10 degrees of further pronation of the forearm (Figs. 2-E and 2-F); and palmar-dorsal [9,17], with the forearm in the semi-supine position—that is, 10 to 30 degrees of supination from the straight lateral position, the ulnar side down and the thumb up and out (Figs. 2-G and 2-H). In all these views the tube-film distance is forty centimeters, and the tube is perpendicular. In addition, the tunnel view (Figs. 2-I and 2-J) may be helpful in problem cases since it brings the carpal canal into profile [8].

FRACTURES OF THE PISIFORM BONE

Thirteen cases were collected from an estimated 6,000 roentgenographic examinations of the wrist carried out in three clinics (Table I). Seven were

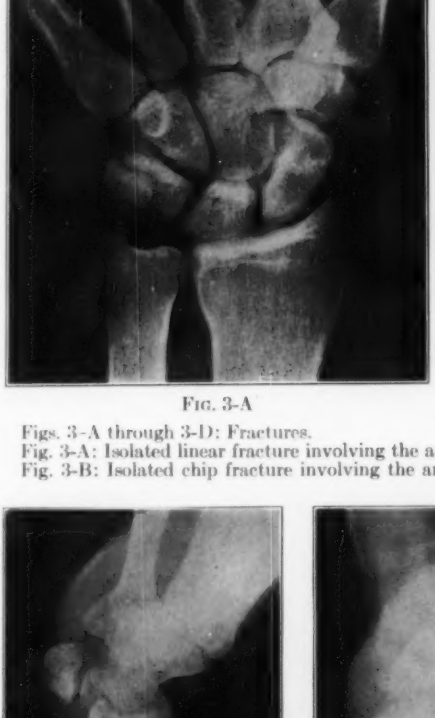

FIG. 3-A

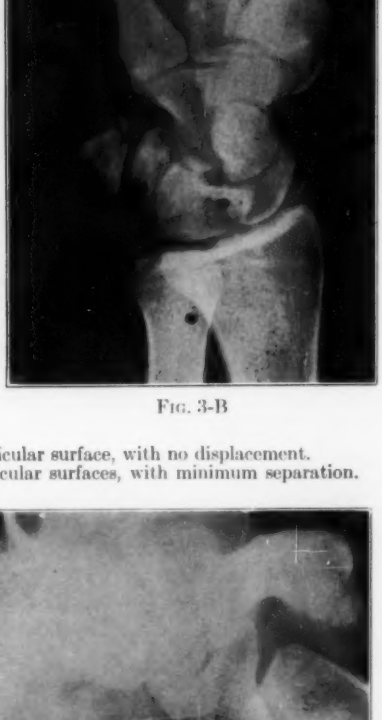

FIG. 3-B

Figs. 3-A through 3-D: Fractures.
Fig. 3-A: Isolated linear fracture involving the articular surface, with no displacement.
Fig. 3-B: Isolated chip fracture involving the articular surfaces, with minimum separation.

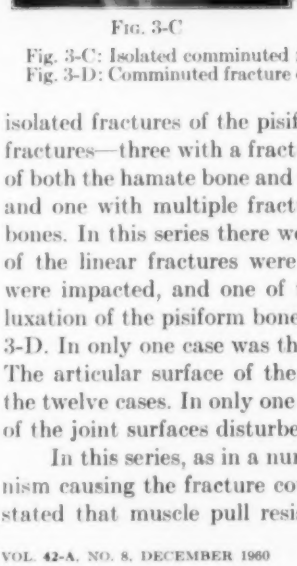

FIG. 3-C

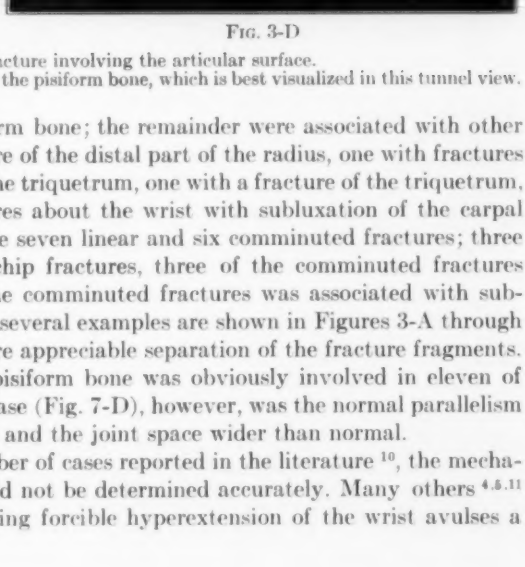

FIG. 3-D

Fig. 3-C: Isolated comminuted fracture involving the articular surface.
Fig. 3-D: Comminuted fracture of the pisiform bone, which is best visualized in this tunnel view.

isolated fractures of the pisiform bone; the remainder were associated with other fractures—three with a fracture of the distal part of the radius, one with fractures of both the hamate bone and the triquetrum, one with a fracture of the triquetrum, and one with multiple fractures about the wrist with subluxation of the carpal bones. In this series there were seven linear and six comminuted fractures; three of the linear fractures were chip fractures, three of the comminuted fractures were impacted, and one of the comminuted fractures was associated with subluxation of the pisiform bone; several examples are shown in Figures 3-A through 3-D. In only one case was there appreciable separation of the fracture fragments. The articular surface of the pisiform bone was obviously involved in eleven of the twelve cases. In only one case (Fig. 7-D), however, was the normal parallelism of the joint surfaces disturbed and the joint space wider than normal.

In this series, as in a number of cases reported in the literature [10], the mechanism causing the fracture could not be determined accurately. Many others [4,5,11] stated that muscle pull resisting forcible hyperextension of the wrist avulses a

FIG. 4-A

FIG. 4-B

Healed fractures.

Fig. 4-A: Same fracture as in Fig. 3-B, four years after injury. The translucent zone at the fracture site suggests fibrous union.

Fig. 4-B: The same fracture as in Fig. 3-C, four years after injury. Note the irregular contour of the joint surface at its distal end. There were no symptoms at this time.

FIG. 5-A

FIG. 5-B

Figs. 5-A through 5-C: Normal kinematics in the semisupine roentgenographic projection (10 to 30 degrees of supination from the straight lateral position).

Fig. 5-A: Neutral position: normal width, parallelism, and symmetry.

Fig. 5-B: Flexion position: increase in the width of the joint space, with maintenance of parallelism and symmetry.

bone fragment. In most of the thirteen cases studied here in which a definite history of the type of injury could be elicited, direct trauma to the pisiform region seemed to be the mechanism.

The isolated fractures of the pisiform were manifested by pain, swelling, and tenderness in the pisiform area, with no associated deformity and very little impairment of motion. In the cases with other associated fractures about the wrist, the signs and symptoms pertaining to the pisiform bone were more or less obscured by the signs and symptoms related to the other injuries.

The treatment of the isolated pisiform fractures consisted in immobilization in a splint or cast for a period of three to six weeks. When fractures of the pisiform were associated with other fractures of the wrist, treatment was directed to the more disabling fracture or injury.

Union of the fractures of the pisiform bone is usually complete in four to six weeks [11]. In one of the cases in this series (Figs. 3-B and 4-A) there was

FIG. 5-C

FIG. 6

Fig. 5-C: Extension position: symmetry, width, and parallelism within normal limits.
Fig. 6: Normal pisotriquetral joint in the neutral position, with width and symmetry maintained with approximately 10 degrees of deviation from parallelism.

apparent fibrous union, as indicated by a persistent translucent zone at the fracture site visible on the roentgenogram made four years after fracture. The patients in this series who have been followed over a sufficiently long period of time (one month to four years) have regained normal function. Traumatic osteoarthritis of the pisotriquetral joint after fracture of the pisiform has been reported by several authors [12,16]. In none of the thirteen patients in this series has this complication developed, but no patient has been followed long enough to exclude this complication.

In the growing pisiform bone, irregularities of ossification may produce confusing bone patterns that not uncommonly present the appearance of numerous bone fragments. After injury, these irregularities may cause difficulty, and comparable views of the opposite wrist are of value with patients below the age of twelve years.

Soft-tissue calcification in the parapisiform area may also simulate chip fractures, but, usually, the intact contour of the pisiform bone and the amorphous pattern of calcium deposit, as opposed to the normal trabecular pattern in the fragment of a chip fracture, make differentiation not too difficult.

The first case of pisiform fracture with roentgenographic evidence that we found in the literature was reported in 1911 by Deane. Since then, other isolated case reports have appeared [4,10,11,13,18]. Stack, in 1938, listed three cases in a review of fractures of the wrist, and Anderson, in 1944, listed four cases from a series of seventy-nine carpal fractures. Böhler, in his book, listed twenty-four pisiform fractures from a total of 826 carpal injuries collected in a ten-year period. The reported frequency of fractures of the pisiform bone, as compared with fractures of other carpal bones, has varied from a ratio of 1:123 [2] to a ratio of 1:170 [20].

ABNORMALITIES OF THE PISOTRIQUETRAL JOINT

In several roentgenograms of the wrist made after injuries, the joint space of the pisotriquetral joint appeared to be abnormally wide, and the normal parallelism of the joint surfaces was lost. Roentgenographically, these appeared to be subluxations, but correlation of these roentgenographic findings with the clinical findings was somewhat unsatisfactory because there were usually associated wrist injuries that tended to obscure the signs and symptoms referable to the lesion of the pisotriquetral joint. Most of these apparent subluxations were

associated with fractures of the distal part of the radius. However, there was one subluxation without associated injury to the wrist that was demonstrated on roentgenograms made after a fall on the hand. This patient had pain and tenderness about the ulnar aspect of the wrist that subsided after immobilization of the wrist in a volar splint for three weeks.

To investigate the significance of these changes, a roentgenographic study of the kinematics of the normal pisotriquetral joint was undertaken, and the roentgenograms of as many fractures of the distal part of the radius as were available were reviewed in an effort to determine the incidence and types of change in the pisotriquetral joint occurring in association with this fracture.

NORMAL PISOTRIQUETRAL JOINT AND ITS KINEMATICS

Motion of the pisotriquetral joint was studied in forty-eight normal adult wrists of twenty-seven male patients and twenty-one female patients. Each wrist was examined roentgenographically in full flexion, in full extension, and in the neutral or anatomical position with the forearm in all instances in the semisupine position—that is, supinated 10 to 30 degrees from the straight lateral position, as illustrated in Figures 2-G and 5-A through 5-C. From these roentgenograms, observations were made with reference to the maintenance of parallel-

TABLE II

ANALYSIS OF THE ROENTGENOGRAPHIC STUDY OF THE KINEMATICS OF THE NORMAL ADULT PISOTRIQUETRAL JOINT IN FORTY-EIGHT CASES

Position of the Wrist Semisupine, 10° to 30° from Lateral Position	Number of Cases		Parallelism of Joint Surfaces		Width of Pisotriquetral Space (Under 3 mm.)		Symmetrical Apposition of the Articular Surfaces	
	Male	Female	Male	Female	Male	Female	Male	Female
Neutral	27	21	25	17	26	21	24	20
Flexion	25*	18*	17	10	15	13	4	3
Extension	24*	19*	8	7	21	18*	3	2

* In the remaining cases the pisotriquetral joint was not satisfactorily visualized.

ism of the articular surfaces, the width of the joint space, and the proximal and distal displacement of the articular surface of the pisiform bone with reference to the articular surface of the triquetrum, or, as termed here, the symmetrical apposition of the two articular surfaces. The findings are tabulated in Table II. In only forty-three of the forty-eight wrists studied could this joint be satisfactorily evaluated in the positions of full flexion and full extension.

Parallelism

Parallelism was determined by observing the relationship of the articular surfaces of the pisiform bone and the triquetrum to each other. In the neutral position, twenty-five of the twenty-seven normal male wrists and seventeen of the twenty-one normal female wrists showed parallelism. In the wrists in which the joint surfaces were not parallel in the neutral position, the degree of deviation from parallelism was 15 degrees or less (Fig. 6). In flexion and extension, the loss of parallelism became more pronounced, amounting to as much as 50 degrees. However, approximately 30 to 60 per cent of the joints retained parallelism of the surfaces in extension or flexion, respectively.

Width of the Joint Space

In the neutral position, the width of the joint space ranged from 1.0 to 4.0

millimeters. In approximately 85 per cent of the cases, the width of the joint space was 2.0 millimeters or less; in only one case was it 4.0 millimeters. Therefore, the upper limit of the normal width of the joint space was accepted as 3.0 millimeters.

Normally, the joint space widens in the fully flexed position, although, as can be seen in Table II, in the majority of cases the width of the space was still within the 3.0-millimeter range with the wrist fully flexed. The maximum width attained in this position was 7.0 millimeters in male patients and 4.0 millimeters in female patients. This moderate increase in the width may be due to looseness of the capsule.

In the extended position, the joint space decreased in width as compared with the neutral position. In all cases the width was less than 3.0 millimeters, and in the overwhelming majority it was less than 2.0 millimeters. This change is presumably the result of tautness of the surrounding collateral ligaments or tightening of the joint capsule associated with wrist extension.

TABLE III

EVALUATION OF THE PISOTRIQUETRAL JOINT IN SEVENTY-FIVE CONSECUTIVE CASES OF FRACTURES OF THE DISTAL PART OF THE RADIUS

Roentgenographic Finding	No. of Cases		
Unsatisfactory visualization of joint	35		
Satisfactory visualization of joint	40		
Normal joint		28	
Abnormal joint		12	
Space wide and surfaces not parallel			6
Space wide and surfaces parallel with lack of symmetrical apposition (20 to 50 per cent overriding)			5
Space normal and surfaces parallel with lack of symmetrical apposition (50 per cent proximal overriding)			1

Alignment or Symmetrical Apposition

The column of Table II, headed Symmetrical Apposition of the Articular Surfaces, records the number of wrists in male and female patients in which the articular surfaces of the pisiform and triquetrum were in exact alignment or symmetrical apposition with no proximal or distal displacement of the width of the articular surface of the pisiform. As can be seen from the table, the overwhelming majority of these normal cases showed symmetrical apposition in the neutral position. Several of the wrists, however, showed from 5 to 10 per cent proximal overriding of the pisiform with the wrist in neutral position. It is concluded, therefore, that slight asymmetry in the position of the two articular surfaces does not indicate subluxation.

In flexion and extension of the wrist, as might be anticipated, there was considerable displacement of the pisiform with reference to the triquetrum. Only a small percentage of the cases showed symmetrical alignment in these positions. Overriding in extension was in the distal direction and ranged from 0 to 50 per cent of the width of the articular surface of the pisiform bone, in the majority of cases ranging from 10 to 30 per cent. In flexion, overriding in the proximal direction was as much as 90 per cent, with almost complete loss of apposition of the two articular surfaces, but in the majority of cases the proximal overriding ranged from 10 to 30 per cent.

Since the normal pisotriquetral joint is often unsatisfactorily visualized roentgenographically in flexion and extension, and, since there is marked deviation of the joint space as to parallelism, width, and alignment of the articular surfaces

FIG. 7-A

FIG. 7-B

Figs. 7-A through 7-E: Subluxation at the pisotriquetral joint.
Fig. 7-A: Widening of the pisotriquetral joint, with maintenance of parallelism and symmetry, associated with displaced comminuted fracture of the distal end of the radius.
Fig. 7-B: Widening of the pisotriquetral-joint space, with loss of parallelism and asymmetry of the joint surfaces, associated with impacted, minimally displaced fracture of the distal end of the radius and fracture of the ulnar styloid process.

FIG. 7-C

FIG. 7-D

Fig. 7-C: Widening of the pisotriquetral joint, associated with undisplaced fracture of the distal end of the radius.
Fig. 7-D: Widening of the pisotriquetral-joint space and loss of parallelism associated with isolated comminuted fracture of the pisiform bone (not well visualized in this projection).

in these positions, only the neutral position can be used to evaluate the condition of the pisotriquetral-joint space after injuries. The problem of subluxation, therefore, can only be discussed with these points in mind.

ABNORMAL PISOTRIQUETRAL JOINT

The roentgenograms of seventy-five consecutive fractures of the distal end of the radius (Colles') were reviewed (Table III). Only forty of these were adequate for the evaluation of the condition of the pisotriquetral joint according to the considerations just enumerated. In twenty-eight of these forty fractures the pisotriquetral joint was normal as to parallelism, width of the joint space, and

symmetry. In twelve cases there was some alteration of the joint space (Figs. 7-A through 7-E); in eleven, the width of the space was more than the normal 3.0-millimeter limit, with a range of 4.0 to 6.0 millimeters, and, in one, the width of the space was normal but the articular surface of the pisiform was displaced proximally 50 per cent of its width. Six joints retained the parallelism of their joint surfaces, and six did not. Five of the joints with parallel surfaces but abnormal width of the joint space showed 20 to 50 per cent overriding of the surfaces. There was no correlation between the severity of the fractures of the distal part of the radius and the condition of the pisotriquetral joint. Since most of the fractures of the distal part of the radius were impacted with resultant shortening of the radius, it is conceivable that the capsular tissues were thereby relaxed sufficiently to cause these abnormalities of the pisotriquetral-joint space. However, in the majority of fractures of the distal end of the radius that were reviewed, the pisotriquetral joint was normal. Hence, it seems unlikely that laxity of the capsule is the cause of joint abnormality.

FIG. 7-E

Widening of the joint space and loss of parallelism and isolated subluxation of the pisiform bone, with no associated fracture (the patient fell on this hand).

In only one of the thirteen fractures of the pisiform bone was there any abnormality of the joint. In view of this, a joint effusion or hemarthrosis is probably not a common cause of these changes in the pisotriquetral joint. Some alteration in the joint capsule or change in the direction of muscle pull on the pisiform bone must be responsible for most of these subluxations.

The patients with subluxations of the pisotriquetral joint were followed up with both clinical and roentgenographic examinations. In one case of subluxation without associated injuries, the joint was normal after three weeks of immobilization. In other cases the joint had returned to normal at the time of follow-up examination. Apparently, the treatment provided for the fracture of the distal part of the radius, including manipulative reduction and immobilization, was sufficient to restore the pisotriquetral joint to normal. In the wrists that did not return to normal, some abnormalities were found to persist. These wrists did not cause symptoms at the time of follow-up examination.

In view of the fact that the clinical findings and symptoms referable to the pisiform area are seldom noted because they are obscured by the signs and symptoms of the more severe associated injuries to the radiocarpal region, the presence of subluxation of the pisotriquetral joint can only be established on the basis of the roentgenographic findings. One or more of the following were considered to be necessary criteria for the roentgenographic diagnosis of subluxation

of the pisotriquetral joint, provided the roentgenograms are made with the wrist in the neutral position and the joint is satisfactorily visualized: (1) a joint space more than 4.0 millimeters in width; (2) loss of parallelism of the joint surfaces greater than 20 degrees; (3) and proximal or distal overriding of the pisiform amounting to more than 15 per cent of the width of the joint surfaces.

From this study it would appear that subluxation of the pisotriquetral joint is of clinical significance when associated with fractures of the distal part of the radius.

SUMMARY

The anatomy and roentgenography of the pisiform and parapisiform area are briefly reviewed. Thirteen cases of fractures of the pisiform bone are reported and discussed, and the scant literature pertaining to this subject is reviewed. The problem of subluxation of the pisotriquetral joint is analyzed on the basis of the normal kinematics of this joint as revealed by roentgenography, and the findings in the pisotriquetral joint in a group of fractures of the distal part of the radius are reviewed.

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Unusual Case of Multiple Abnormalities of Bone with Malignant Degeneration

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This report concerns a bone disease that is different from any of the commonly recognized lesions of bone. The pathological changes found in this case include some of those found in fibrous dysplasia, multiple enchondromatosis, and hemangiomatosis of bone. Some of the lesions in this case finally evolved into osteogenic sarcomata. It is remarkable, perhaps, that dysplasias of bone do not more commonly involve these many bone elements.

FIG. 1

FIG. 2

Fig. 1: Lateral roentgenogram of the facial bones made when the patient was eleven years old. Note numerous bone masses beneath the jaw, some of which appear to be fused to the inferior margin of the right mandible.

Fig. 2: Anteroposterior and lateral views of the arm when the patient was eleven years old, showing the tumor. The outline of the carpal bones is completely lost except for a few clefts suggesting joint spaces. The distal ends of the radius and ulna and proximal ends of several metacarpals are also involved.

CASE REPORT

J. P., L. H. #23921, a Negro boy, was first seen at Lincoln Hospital, Durham, North Carolina, when he was eleven years old. He was admitted for removal of tumors involving the left wrist and mandible. The tumor involving the wrist had first been noted when the patient was five years old, and that of the jaw when he was nine years old. It could not be definitely determined whether the abnormality of the left wrist had existed before the child was five years old, but the jaw lesion had definitely appeared in his ninth year. A biopsy of the lesion in the left wrist had been performed at another hospital in the child's sixth year, but this material could not be located for review, and there was no available pathological report. The lesions of the wrist and jaw had slowly enlarged until the child's eleventh year.

FIG. 3

FIG. 4

Fig. 3: Roentgenogram of the left arm and hand when the patient was twelve years old, one year after the removal of the tumor and the insertion of a tibial graft. Note the fusion of the graft to the radius and metacarpals and the irregular masses of bone at the operative site.

Fig. 4: Roentgenograms of left arm and hand when the patient was sixteen years old, three weeks prior to death. Note the abundant irregular new bone about the wrist completely filling the defect left by resection of the distal end of the ulna.

FIG. 5

Fig. 5: Roentgenogram of pelvis when the patient was sixteen years old, three months prior to his death. Note the ossifying tumor and the soft-tissue mass just lateral to the right ilium, above the acetabulum. Note irregular densities in each femur along the intertrochanteric line, more on the left.

On his first admission to the Lincoln Hospital, the old operative scar was noted over the left wrist, overlying a palpable tumor that had fixed the wrist joint in flexion and had limited the motion of the thumb. The mass at the chin was located along the inferior margin of the right ramus of the

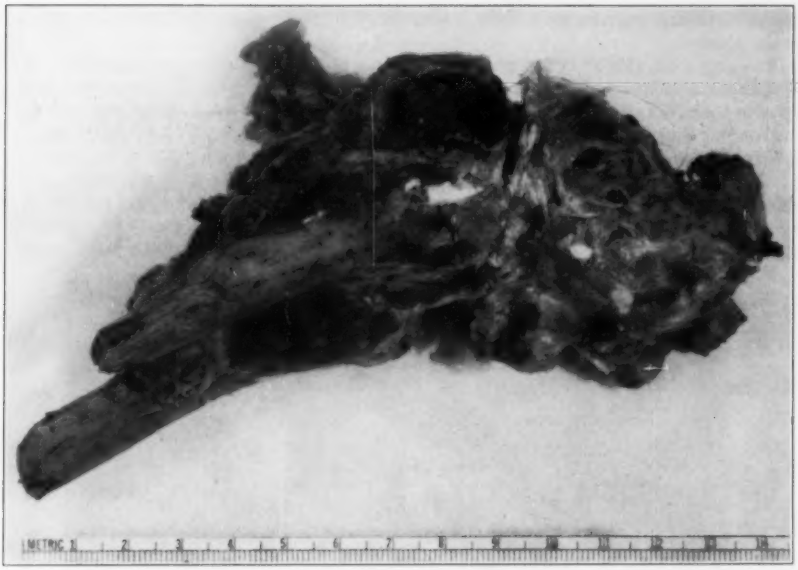

FIG. 6

Fig. 6: Gross specimen of the tumor removed from the left wrist when the patient was eleven years old. The ends of the radius and ulna (left) and carpal bones (right) are lost in a fibrous and bone mass at the center.

mandible and was continuous with the mandible. The remainder of the physical examination was not remarkable.

The blood inorganic phosphate was four milligrams per 100 milliliters, the calcium was twelve milligrams per 100 milliliters, and the alkaline phosphatase was 7.7 Bodansky units. The Kahn test was negative. The red-cell count was 3.9 million, and the white-cell count was 5,000 per cubic millimeter, with a hemoglobin of eleven grams per 100 milliliters. Roentgenograms revealed that the left wrist was markedly deformed by the tumor with ulnar and volar flexion (Fig. 2). The carpal bones were fused into a mass of sclerotic bone, with obliteration of all the carpal joints. The lesion also involved the distal end of the radius and the ulna and the bases of the metacarpals of the thumb and index and long fingers. Roentgenograms of the right mandible showed a large bone mass, five by ten centimeters, extending along the inferior margin of the mandible from the angle to the symphysis. This tumor was composed of multiple irregular sclerotic bone segments (Fig. 1). There were no dental deformities. Other scout films of the skeleton showed a cortical lesion of the left humerus and a sclerotic lesion in the superior margin of the right ilium just above the acetabular brim. In addition, there were bone densities in the proximal portions of both femora, more on the left, that were thought to be abnormal in 1952 and 1957. The lesions involving the humerus and femora were described as being consistent with sclerosing hemangiomata. Subsequent to these examinations in 1952, the lesion in the humerus remained unchanged on clinical examination.

On February 11, 1952, a right submandibular incision was made, exposing an encapsulated nodular bone mass that was attached to the mandible. This mass extended downward almost to the level of the right lobe of the thyroid. A plane of dissection from the mandible was found, and the tumor was separated from the mandible with ease. All visible tumor was removed. The excised tumor was found to be a large, firm, bone mass composed of multiple pieces of well formed bone of irregular contour, ranging in size from two millimeters to four centimeters with some fibrous and cartilaginous tissue attached; some smaller bone masses were loosely attached to the main mass by fibrous tissue. These masses of bone corresponded to the pebble-like masses observed in the roentgenogram and were separated from each other and from the mandible by sheets of dense collagenous tissue with some looser areas containing blood vessels and fat (Fig. 13). The sheets of collagenous tissue were split in a few areas in such a way as to suggest synovial-lined spaces, although synovial tissue could not be identified. Numerous small spicules of new bone surrounded by prominent osteoblasts were found in the collagen. The outermost layer of the bone had a dense periosteum from which osteoid and bone were laid down directly. Larger trabeculae were found in the central

portions of these bones; between these trabeculae were areas of hemorrhage and rare dilated vascular channels.

Recovery from this operation on the mandible was uneventful. The patient re-entered the hospital four months later for resection of the tumor of the left wrist. At this second hospital admission there was no recurrence of the mandibular lesion, and the surgical scar was healed. The left wrist was again described as being deformed by an irregular tumor, with the hand held in 90 degrees of flexion and 25 degrees of ulnar deviation. There was no motion in the wrist. At this time his red-cell count was 4.2 million, his white-cell count was 4,200 per cubic millimeter, and the hemoglobin was 14 grams per 100 milliliters. Roentgenograms were again made of the wrists, but they showed no appreciable change from those made four months before, except for the development of what appeared to be slight involvement of the terminal phalanx of the index finger. At

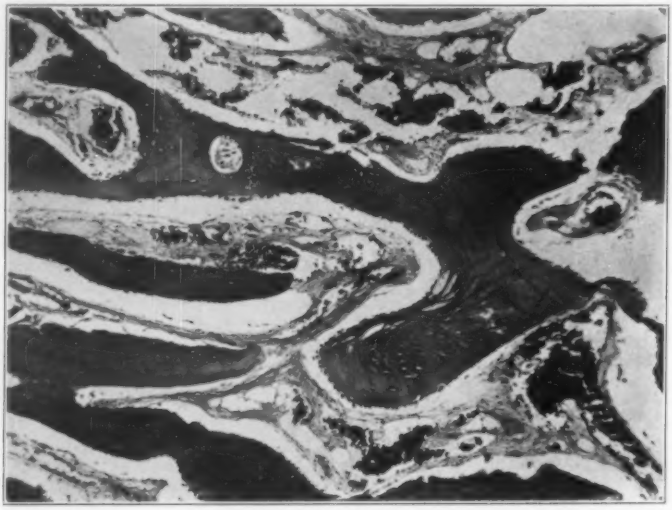

FIG. 7

Vascular hemangiomatous channels between bone trabeculae. (Radial lesion; hematoxylin and eosin, × 65.)

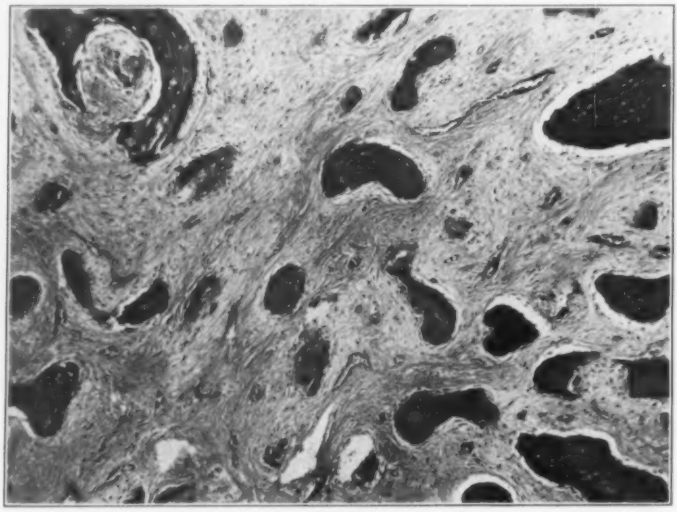

FIG. 8

Islands of bone in a dense fibrous stroma, simulating fibrous dysplasia. (Radial lesion; hematoxylin and eosin, × 65.)

surgery on June 26, 1952, a longitudinal incision was made over the dorsum of the forearm and wrist, and the entire mass of tumor was easily separated by extraperiosteal dissection from the surrounding tissues. Proximally, the radius and ulna, and, distally, the thumb, index, and long metacarpals were sectioned subperiosteally through normal bone, and the tumor was excised in one block. The mass removed included approximately the distal third of the radius and ulna plus two-thirds of the first, second, and third metacarpals (Fig. 6). The tumor was firm, fibrous, and bony, and of irregular contour; it did not appear grossly to infiltrate surrounding tissues. The major nerves and vessels were preserved in this dissection, but the extensor tendons were thin, difficult to detect, and were destroyed in some instances. A tibial cortical graft approximately eight inches long was taken and fixed by wire loops to the second and third metacarpals and by three screws to the radius. The postoperative course was uncomplicated. The pathological specimen

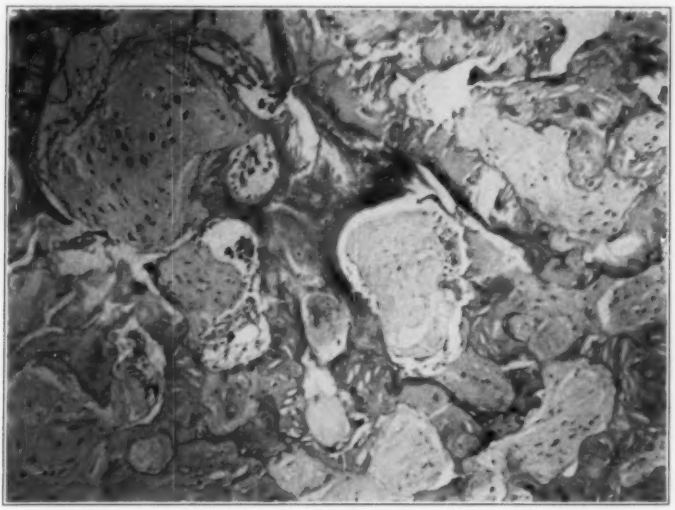

FIG. 9

Irregular masses of abnormal cartilage within bone. (Radial lesion; hematoxylin and eosin, × 110.)

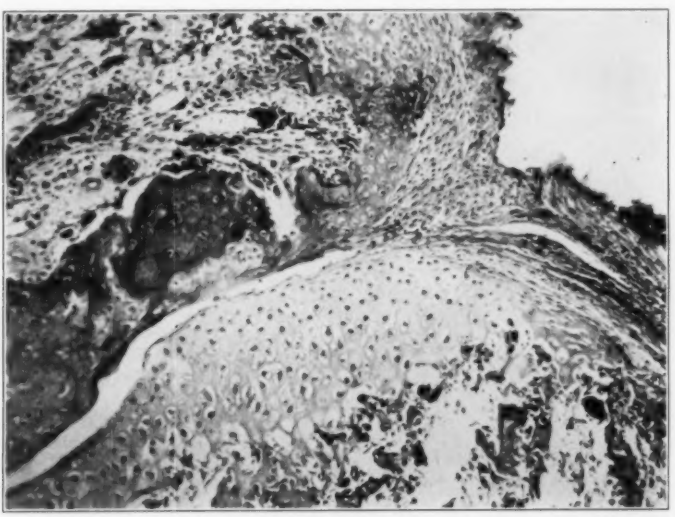

FIG. 10

Cystic space lined by cartilage in the radial lesion; this is probably the vestige of a joint space. (Radial lesion; hematoxylin and eosin, × 110.)

included the distal ten or fifteen centimeters of the radius and ulna, the conglomerate tumor involving the distal ends of the radius and the ulna, the obliterated radio-ulnar and radiocarpal joints, the carpal bones and intercarpal joints, the carpometacarpal joints, and the proximal ends of the first, second, and third metacarpal bones. In the conglomerate mass, in the region of the carpus, the individual bones could not be distinguished. The mass was composed of bone covered by dense collagenous tissue. Some hemorrhagic and edematous tissue surrounded the ends of the radius and the ulna. A broad section extending from the proximal to the distal end of the specimen was taken for microscopic study. Even by this means, individual bones could not be recognized. Some of the joint spaces were represented by dense collagenous bands and spaces with a thin zone of cartilage in some places (Fig. 10). The masses of tumor were composed of bone trabeculae and fatty marrow and some irregular cartilaginous masses (Fig. 9). Broad strips of cartilage, probably

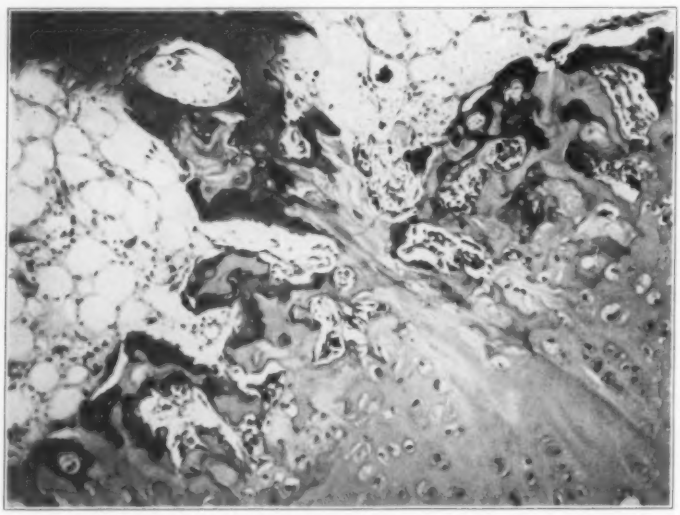

FIG. 11

Ossification from cartilage at the margin of an endosseous mass of cartilage, resembling an epiphyseal line. (Radial lesion; hematoxylin and eosin, × 110.)

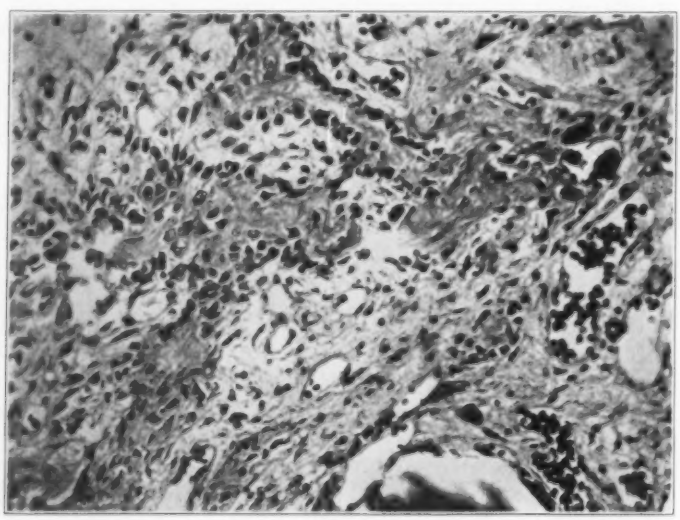

FIG. 12

Osteoid with surrounding osteoblasts. (Radial lesion; hematoxylin and eosin, × 235.)

representing abnormal joint cartilage or displaced epiphyseal plates, coursed in a seemingly aimless fashion through bone, and enchondral ossification was proceeding along margins of cartilage (Fig. 11). Adjacent to the cartilaginous masses and separating some of the masses of bone were broad fibrous areas in which small fragments of new bone were forming (Fig. 12). In some larger fibrous areas within the tumor, there were multiple small islands of bone exactly similar to those found in fibrous dysplasia (Fig. 8). In some portions of the tumor there were large cavernous thin-walled vascular channels that contained blood and were backed against each other (Fig. 7). Relatively little fibrous tissue was associated with these hemangiomatous area, which were encompassed by an outer zone of dense cortical bone.

Roentgenograms made on September 2, 1952, showed that the graft had united with the radius and with the metacarpals of the index and long fingers. At this time, the fingers were freely movable

FIG. 13

Two of the extramandibular masses of new bone with intervening fibrous and collagenous tissue; bone is forming directly from the periosteum. (Jaw lesion; hematoxylin and eosin, × 65.)

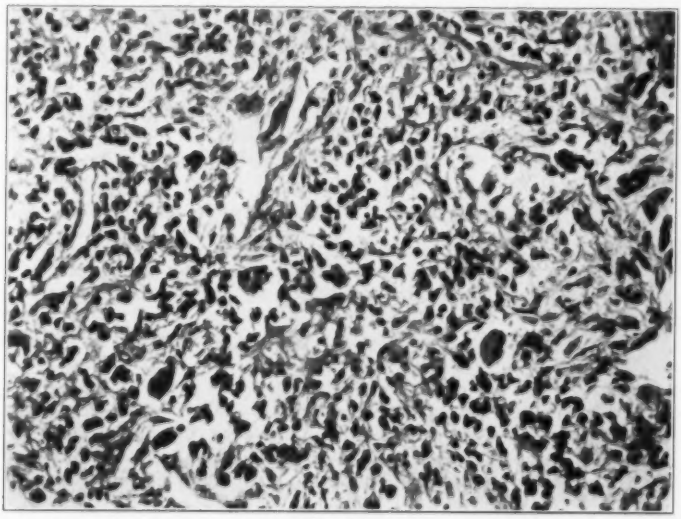

FIG. 14

Osteogenic sarcoma from the right hip. Note pleomorphism of tumor stromal cells and the tumor giant cells; small masses of osteoid can be seen. (Hematoxylin and eosin, × 65.)

and had a good range of motion without contractures. The large graft was gradually revascularized and, by March 3, 1953, it had been replaced with the new bone (Fig. 3). In addition, new bone was present on the interosseous membrane at the proximal third of the forearm and at the severed end of the ulna. For some distance distal to the severed end of the ulna, additional new bone had grown from periosteal remnants, seeming to join with the radius at this point by a tumor-like mass of bone (Fig. 3).

The patient was readmitted to Lincoln Hospital on August 8, 1955, at the age of fourteen years, for multiple tendon transfers about the left hand. The sublimis tendons to the index, ring, and long fingers were transferred to the extensor mechanism of the index, ring, and long fingers and to the extensor pollicis longus.

Two years later the patient was readmitted to the hospital on August 9, 1957, complaining of pain of six weeks' duration in the right hip and buttocks that radiated into the right leg. There had been no loss of appetite or loss of weight. The face was somewhat asymmetrical and had been so without clinical change since shortly after operation. The right lower jaw was more prominent than the left, and palpation of the right lower jaw revealed it to be thicker than the left. The left forearm was about four inches shorter than the right; it was very irregular in contour due to abnormal new-bone prominences in the area of the graft. The ends of the radius and ulna and the remaining portions of the metacarpals on the left hand fused with the bone graft, and a new mass of bone filled the defect left by previous excision of the ulna (Fig. 4). The region of the right hip joint and just above the joint seemed swollen and hard. This region was larger and more prominent than the same area on the left, and the patient limped on the right side. A roentgenogram of the pelvis showed an ossifying tumor just lateral to the right ilium and a soft-tissue mass (Fig. 5). The chest roentgenogram revealed no abnormality at this time. A biopsy of the right iliac tumor was performed on August 9, 1957. It was a large tumor involving not only the periosteum and cortex of the right ilium, but also the overlying muscle and fascia. There was complete destruction of all normal bone and extension of the tumor outward from the normal limits of the ilium for about an inch as far as the gluteus medius muscle. The bone and soft-tissue tumor measured about three by three by three inches and extended from an area one and one-half inches below the iliac crest to within an inch of the acetabular brim. The tissue obtained at biopsy included multiple irregular pieces of fibrous, dark-brown, grey, and light-brown tissue measuring up to 4.5 centimeters in length. Microscopically, the tumor was composed of closely packed large vesicular cells of varying size. Some of these cells were producing a light-pink osteoid matrix. A number of large multinucleated giant cells were scattered throughout this lesion, and there were many mitotic figures. The histological diagnosis was osteogenic sarcoma, osteolytic type. (This tumor was identical in microscopic appearance to the tumor in the ilium and wrist found later at autopsy.) The patient was discharged August 21, 1957, and readmitted on October 19, 1957, in a terminal state with pain and bleeding in the right hip. New roentgenograms of the left arm showed much new bone in the wrist and forearm in the region where the tumor had been resected and replaced by a bone graft (Fig. 4). A large, friable, fungating mass was present at the right hip and bled profusely when the dressing was removed. Throughout his hospital stay the patient continued to complain of pain in the hip. His hemoglobin at this time was 4.5 grams per 100 milliliters. On November 3, 1957, he suddenly became somewhat more comfortable and was discharged since he was anxious to return to his family. He died in his sixteenth year on November 9, 1957, and an autopsy was performed at Lenoir Memorial Hospital, Kinston, North Carolina *.

POST MORTEM EXAMINATION

The body measured more than six feet in length and appeared fairly well nourished. The lower extremities were edematous. There was a large, ulcerated, sloughing, grey tumor that involved the entire area of the right lateral hip and measured thirty to forty centimeters in its greatest dimensions. The necrotic and hemorrhagic tumor involved the ilium, with erosion of the hip joint and upper portion of the femur. The mass also protruded through the necrotic and infected overlying skin. A long operative scar extended the entire length of the left forearm from the elbow down to the hand. A portion of the radius and bone from the graft area were removed. A bone mass was palpated at the level of the left wrist and the proximal ends of the radius and ulna, and the graft fused to the wrist mass could also be felt. The contour of the bones at the wrist was irregular, but no soft-tissue invasion was recognized. The right mandible appeared larger than normal. No portion of the mandible was taken for study because the family objected. There was a healed surgical scar beneath the mandible. Other bones were not examined. A pint of free fluid was found in the peritoneal space. There were dilatation of all chambers of the heart and edema of the lungs.

* The authors are indebted to T. S. Wedde, M.D., pathologist and prosector, for the description of the gross autopsy and for the tissues for microscopic examination.

The patient had had a severe anemia, and it was thought that this contributed to heart failure, the immediate cause of death. There was no evidence of metastatic tumor in any of the thoracic or abdominal organs. Tumor tissue was found only in the bones and in the connective tissue and muscles immediately adjacent to the bones. This finding makes it probable that the bone tumors were multicentric in origin, rather than metastases arising from one primary lesion.

Tissue from the tumor of the right hip was non-gritty, white, and firm. It contained soft areas of necrosis and areas of viable tumor. Microscopically, the tumor was composed of a solid sheet of fusiform and polyhedral cells with large nuclei and clumped chromatin, suggesting a very cellular fibrosarcoma of bone. Mitotic figures and some multinucleated giant cells were seen. Small masses of osteoid with early ossification separated tumor cells in a few areas (Fig. 14). Tumor penetrated the cortex of the ilium into the periosteum and adjacent collagenous tissue and muscle.

Tissue taken from the wrist was gritty and contained some dense cortical bone. In some areas there were normal bone trabeculae and fatty marrow, but elsewhere the bone was infiltrated and replaced by sheets of small spindle tumor cells. This tissue was histologically identical to that from the hip lesion, with both lesions probably representing osteogenic sarcomata of multicentric origin.

DISCUSSION

The important lesions of bones originally seen in the roentgenograms of this patient involved the metacarpal and carpal bones of the left hand, the distal ends of the left radius and ulna, the right jaw, and the right ilium. There was a very unusual obliteration of the joints between the various metacarpal and carpal bones and between the carpal-bone mass and the radius and ulna. In addition to displaced masses of cartilage, there were areas resembling fibrous dysplasia and still other areas containing multiple cavernous hemangiomata in the wrist lesion. The obliteration of the joints at the wrist was apparent in the roentgenogram as well as in the gross specimen and may be a sign of some use in distinguishing this disorder. There were multiple small new bones formed in the soft tissue about the right mandible, an appearance very different from the fusiform swelling characteristic of most tumors or tumor-like conditions of bone. The wrist lesion was first noted when the patient was five years old. No lesion was known to be associated with trauma or other exogenous agents. Osteogenic sarcomata finally appeared in the patient's sixteenth year in the ilium and wrist, probably being multicentric in origin. Hence, this disorder involved multiple bones of a child and was characterized by the obliteration of joint spaces, the formation of new bones, a histological picture of jumbled multiple bone elements, and final sarcomatous degeneration.

Some aspects of this disease are quite similar to other bone diseases. The polyostotic involvement, the age of the patient, and, to some extent, the roentgenographic appearances and histological features of the bone lesions suggest polyostotic fibrous dysplasia. The vascular channels found between the bone trabeculae in the wrist lesion of this case resemble those found in either a solitary hemangioma [3,7,8,19] or in multiple hemangiomata [8,17]. Furthermore, osteogenesis or sclerosis somewhat similar to that seen in some areas of this lesion has been described in hemangiomata [2,8,11,14]. The enchondral cartilaginous masses and the age of the patient are suggestive of enchondromatosis [6,15]. In Maffucci's syndrome enchondromatosis is associated with hemangiomata [4,15]. However, the hemangiomata in Maffucci's syndrome, unlike those in this case, are in the soft tissues and not in bone [4,5,12,18]. Osteogenic sarcoma may complicate Paget's disease, as it complicated this disorder [10]. Fibrosarcoma of bone, osteogenic sarcoma, and chondrosarcoma may rarely complicate fibrous dysplasia, and chondrosarcoma complicates about 50 per cent of cases of multiple enchondromatosis [10]. Sarcoma has not been reported to complicate hemangiomatosis.

Despite similarities, there are many differences between the disorder we describe and these entities just mentioned. The prominent multiple carti-

laginous masses, new-bone formation, large areas of fatty marrow, cavernous hemangiomatosis, and obliteration of joint spaces are not characteristic of fibrous dysplasia; the cartilage and joint abnormalities are quite foreign to hemangioma of bone. The lesions in multiple enchondromatosis tend to be unilateral; the lesions in this case were not. The cartilage in our case resembled normal epiphyseal cartilage with respect to nuclear population and type, whereas some hypernuclearity and atypia is more usual in multiple enchondromatosis. New-bone formation from cartilage and from fibrous tissue resembling fibrous dysplasia is not characteristic of enchondromatosis.

Rare reports of cases of bizarre bone disease that have not seemed to fit into presently accepted classifications appear in the literature. Examples of these cases that we have reviewed differ from our case in some important aspects. For instance, the case of Blumberg, Dehne, and Forbus possesses none of the well defined cartilaginous masses observed in our case. The osteoid lesion described by Jaffe and Mayer and later named benign osteoblastoma by Jaffe similarly had no cartilage. Lichtenstein's description of twenty-five unusual chondroid tumors does not include any description of lesions similar to those found in our case [13].

If, in the future, other cases are found similar to this one with the same distinctive morphological combination and the tendency to develop into sarcoma, there will be little doubt of the need for a separate term for this entity. The term polymorphous dysplasia seems to us to embody the abnormal growth and development of bone and the complex histological features that characterize this lesion.

SUMMARY

A bone disease different from any disease commonly recognized was found in one patient observed by us. The pathological changes were a combination of those found in fibrous dysplasia, multiple enchondromatosis, and hemangiomatosis, with final evolution into osteogenic sarcoma.

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(Continued on page 1348)

Further Studies on Experimental Spondylitis and Intercorporeal Fusion of the Spine

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Since interbody fusion appears to be the almost inevitable result of pyogenic infection involving the intervertebral discs in the human being, it seemed reasonable to assume that interbody fusion might be induced by chemical means. If this process was feasible, means more reliable than those in current use might be available for treating instability of the spine. With this thought in mind, we were interested in determining if interbody fusion similar to that seen in human beings after infection in the intervertebral space could be induced in dogs by inflammatory substances other than live pathogenic bacteria.

In a pilot study reported in 1957 [18], sixteen interspaces in eight dogs were injected with either a solution of 5 per cent sodium psylliate with 2 per cent benzyl alcohol (sylnasol) or a suspension of killed *Staphylococcus aureus*. Three of the sixteen interspaces so injected showed destruction, with the formation of hypertrophic spurs of new bone similar to those observed after clinical infection of the interspace in human beings. It is the purpose of this communication to report further investigations along these same lines.

MATERIALS AND METHODS

Seventy-four adult mongrel dogs were subjected to the surgical introduction of an inflammatory agent into one of the lumbar interspaces. Three types of agent were selected: (1) irritants, (2) proteolytic enzymes, and (3) killed and living pyogenic bacteria. Table I records in detail the material used and the results obtained.

The dogs, seven to twenty kilograms in weight, were operated on with thiopental sodium as the anesthetic. The surgical approach to the disc was by laminectomy in twenty instances, by needle puncture in four, by lateral transversectomy in one, and by an anterior transabdominal incision in forty-nine. The transabdominal approach proved best for the visualization and curettage of the lower lumbar discs. In this procedure, after entering the peritoneal cavity through a mid-line incision, the anterior longitudinal ligament was exposed through an incision in the posterior peritoneum just above and to the left of the aortic trifurcation. The fourth, fifth, or sixth lumbar interspace was entered by cutting a trap door in the ligament with a No. 11 Bard-Parker knife blade. The gelatinous disc material and the cartilaginous plates were curetted thoroughly through this trap door. When the disc space was quite dry, the abdominal contents were covered with an abdominal pack, and three or four drops of the inflammatory agent were instilled directly into the interspace with a syringe and a long No. 20 needle. If possible, the trap door in the ligament was then resutured in place. Occasionally, when this was not possible, a pledget of gelfoam sponge was placed over the window. The posterior peritoneum was not sutured, and the abdominal wall was closed in layers.

* The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

TABLE I

EXPERIMENTAL SPONDYLITIS IN SEVENTY-FOUR DOGS

Materials Placed in the Intervertebral Space	No. of Dogs	No. of Fusions	No. of Deaths	Cause and Time of Death
Irritants:				
Sodium morrhuate, 10 per cent solution	5	0	0	
Talc	2	0	0	
Potassium stearate and potassium oleate	1	0	1	During anesthesia for roentgenography; ten weeks after operation
Croton oil	8	1	2	Chemical peritonitis; 24 and 48 hours, respectively, after operation
Proteolytic enzymes:				
Sterile gastric juice	1	0	0	
Clostridial collagenase	3	0	1	Undetermined; 7 days after operation
Bacteria:				
X-108* (1 million units per milliliter)	1	0	0	
X-108 and urea	4	0	0	
Suspension of killed *Staphylococcus aureus*	23	0	0	
Suspension of killed *Neisseria gonorrhoeae*	3	0	0	
Suspension of live *Staphylococcus aureus*	23	23	1	Pneumonia; 2 weeks after operation
Totals	74	24	5	

* X-108 is an experimental proteolytic enzyme kindly supplied by Mr. Frank Ablondi of the American Cyanimid Company.

After operation, the animals were kept in individual cages and were not immobilized in any fashion. All animals were up and about as soon as the anesthetic had worn off. Roentgenograms were obtained at intervals from four weeks to one year after surgery. Several animals were sacrificed at intervals of one week to one year, and the lumbar portions of their spinal columns were removed for study.

RESULTS

Five animals died after surgery. Two of these deaths occurred twenty-four and forty-eight hours, respectively, after operation and were attributed to peritonitis caused by the spilling of a few drops of croton oil inadvertently in the peritoneal cavity at the time of operation. The third dog died of unknown cause seven days after operation. The fourth dog died of pneumonia two weeks after operation, and the fifth died during anesthesia while a roentgenogram was being made ten weeks after operation. After the first four or five postoperative days none of the surviving animals appeared sick, and in no instance was there evidence of neurological deficit, meningitis, or paraspinal abscess.

As seen in Table I, consistent intercorporeal fusion was obtained only in the group treated with a suspension of live staphylococcal organisms. Among the eight animals treated with croton oil, fusion was obtained in one and was thought to be due to contamination. No cultures were made, however. In this particular instance an effort had been made to fill the interspace with ostamer. When narrowness of the interspace prevented this, the attempt was abandoned, and croton oil was introduced instead. We believe that the needle was contaminated in the removing of croton oil from the bottle. We are now convinced that the fusions reported in the pilot study also were probably due to contamination by

FIG. 1-A FIG. 1-B FIG. 1-C

Fig. 1-A: The sixth lumbar interspace twenty-six weeks after instillation of croton oil. There are no indications of fusion. Minimum narrowing and sclerosis were the only changes observed in all dogs treated with agents other than living staphylococcal organisms.

Fig. 1-B: The fifth lumbar interspace fourteen weeks after instillation of croton oil. The interspace was narrowed, and the adjacent bone was sclerotic, but indications of fusion were lacking. Proliferation of new bone was apparent only along the anterior surfaces of the fifth and sixth vertebral bodies, where the anterior longitudinal ligament was inflamed by croton oil.

Fig. 1-C: The fifth lumbar interspace five weeks after infection with live staphylococci. Note the fuzzy outline of contiguous cartilaginous plates, sclerosis of adjacent bone, and slight increase in the anteroposterior diameters of the bodies of the adjacent vertebrae.

the needle (which probably became contaminated as it passed through the skin) of the interspace at the time of the percutaneous injections.

Most of the animals treated with agents other than live staphylococcal organisms showed slight narrowing of the interspace, and sclerosis of the epiphyseal plates (Fig. 1-A) developed in a few. There was evidence of the formation of new bone along the concave anterior surface of the vertebral body in two of the animals treated with croton oil (Fig. 1-B), but in none of the eight spines treated with this agent were there the pronounced narrowing, erosion, lipping, and hypertrophic change noted in the spaces treated with live organisms.

The roentgenographic changes seen in the animals treated with live staphylococcal organisms resembled those seen in clinical infections of the intervertebral disc. Roentgenograms and gross sections made five weeks after infection (Figs. 1-C and 1-D) showed erosion of the vertebral end plates adjacent to the affected space. At six to eight weeks (Fig. 2-A), progressive sclerosis of bone seemed to involve almost half of the bodies of the adjacent vertebrae, and the interspace itself became narrower, and its margins became irregular. The contiguous halves of the vertebral bodies above and below the involved disc space grew wider, as seen in both anteroposterior and lateral roentgenograms. The formation of this new bone (Figs. 2-B and 2-C) appeared to reach its maximum at eight to twelve weeks after operation, at which time the interspace was almost obliterated, and the large beaks of bone, which had formed anteriorly, began to coalesce. From this point on, the principal change seen in the roentgenogram was a remolding process by which the fusiform bone mass that had formed about the interspace dwindled to a cylinder of bone with approximately the same diameter as that of the normal vertebral bodies (Figs. 3-A and 3-B).

Thus, the entire chain of events after infection with *Staphylococcus aureus*

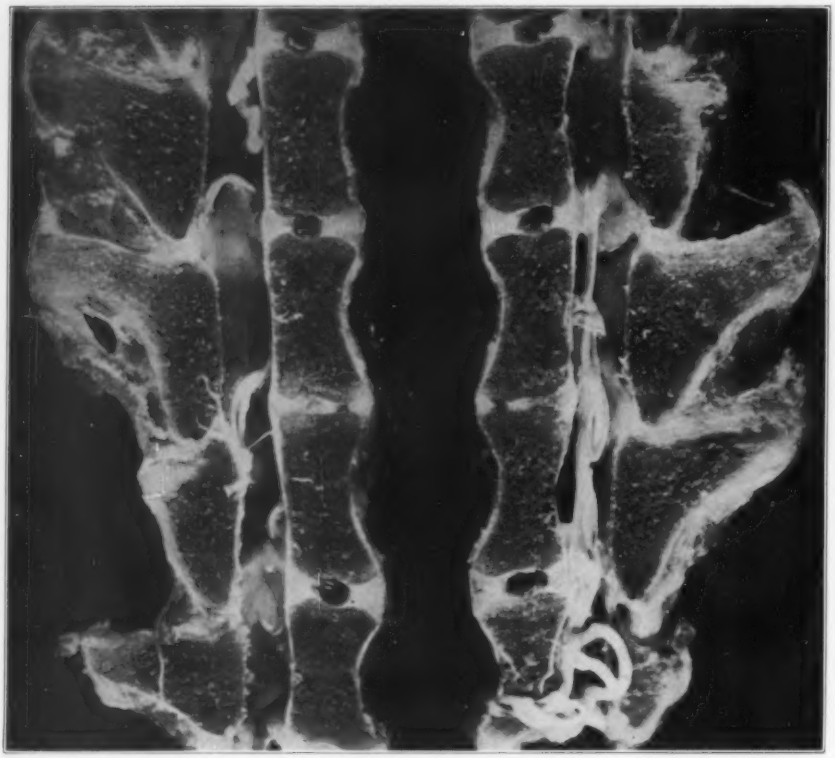


FIG. 1-D

Sagittal section of the same spine as in Fig. 1-C, also at five weeks, showing the narrowed interspace and the irregular outline of the adjacent vertebral surfaces. There is no actual abscess, and there is no gross impingement on the spinal canal.

FIG. 2-A FIG. 2-B FIG. 2-C

Fig. 2-A: The sixth lumbar interspace eight weeks after infection with live *Staphylococcus aureus*. Progressive narrowing of the interspace associated with proliferation of new bone is evident.

Fig. 2-B: Lateral view at nine weeks after infection with live *Staphylococcus aureus*. The formation of new bone appeared most abundant at this time; anterior beaks are beginning to coalesce.

Fig. 2-C: Anteroposterior view (at the same time as in Fig. 2-B), showing abundant formation of new bone and the gradual obliteration of the interspace.

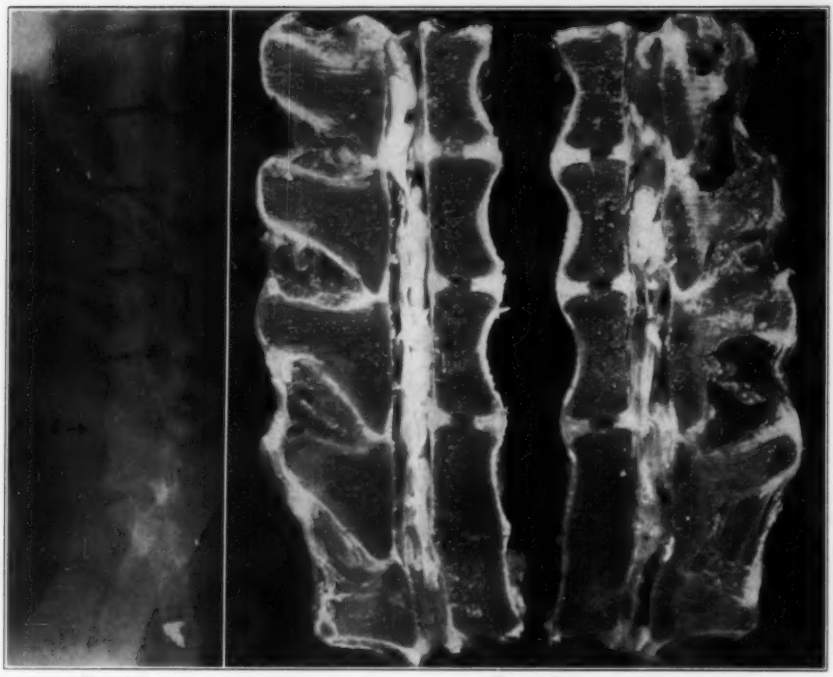


FIG. 3-A FIG. 3-B

Fig. 3-A: Solid intercorporeal fusion of the sixth lumbar interspace twenty-five weeks after the introduction of living *Staphylococcus aureus* through a window in the anterior longitudinal ligament.

Fig. 3-B: Sagittal section of the spine, showing the continuity of cancellous bone across the interspace. The diameter of the spinal canal is not altered. Remodeling has occurred; the bodies are approximately normal in diameter.

falls into three stages: (1) inflammation and destruction, which occur during the first six weeks; (2) proliferation of new bone and repair, which take place from the sixth to the twelfth week; and (3) remolding of the fusion site, which is usually completed at twenty-four weeks.

FACTORS INFLUENCING FUSION

The exact mechanisms involved in spontaneous bony ankylosis of the intervertebral space after pyogenic infection are complex and far from clear. The following factors appear to influence fusion and warrant some discussion.

Surgical Technique

The fact that simple mechanical or surgical destruction of the disc and cartilage plates does not bring about fusion is obvious from our own data and those of others. Compere stated that he had never seen bone fusion in man or animal after curettement of the disc space. Key and Ford, in a study of experimental disc lesions in fourteen dogs, vigorously curetted one lumbar interspace in each animal. Despite removal of the nucleus pulposus and curettage of the cartilaginous plates to bleeding bone, they found no evidence of fusion in any of their animals. Jenkner and his associates obtained "firm fixation" of the interspace in monkeys after evacuation of the disc, curettage of the cartilaginous plates, and insertion of basic calcium-phosphate paste, but their roentgenograms and histological sections do not reveal intercorporeal fusion.

FIG. 4-A

FIG. 4-B

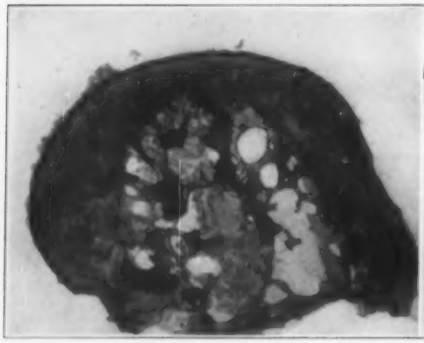


FIG. 4-C

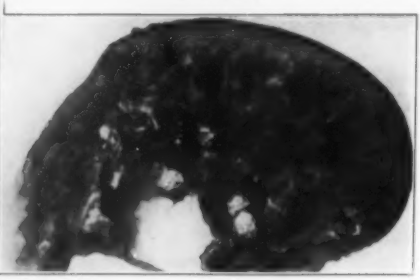


FIG. 4-D

Figs. 4-A, 4-B, 4-C, and 4-D: Distal articular surface of a rabbit's femur. Slices of fresh bone cut on a milling machine to a thickness of 0.04 inch, were immersed in a test solution for twenty-four hours at 36 degrees centigrade.

Fig. 4-A: Control. Section immersed in isotonic saline solution with pH 6.8 phosphate buffer.

Fig. 4-B: Section treated with tryptar and urea.

Fig. 4-C: Section treated with X-108 and urea; note the disappearance of the cartilage surfaces.

Fig. 4-D: Section treated with clostridial collagenase; note the disappearance of the cartilage surfaces.

Motion

Undoubtedly, the degree of immobility of a joint affects to some extent the likelihood of fusion after pyogenic infection. This is suggested by the regularity with which intervertebral joints, apophyseal joints, sacro-iliac joints, and the symphysis pubis fuse, whereas the more mobile joints of the extremities are less likely to fuse. In a series of posterior-element fusions in dogs, Hurley and associates were unable to obtain fusion of the seventh lumbar (lumbosacral) joint, whereas the other lumbar segments fused more readily. As our surgical measures were carried out before their report was published, we utilized only the fourth, fifth, and sixth lumbar segments. Humphries and Hawk were able to obtain solid intercorporeal fusion of the lumbosacral joint in the dog through immobilization by means of an anterior metal plate.

Proteolytic Enzymes

In all probability, proteolytic enzymes play the major role in destroying the intervertebral structures and in providing the chemical environment necessary for the subsequent bone proliferation that leads to interbody fusion. There are three apparent sources of these enzymes in pyogenic infections of the intervertebral space: (1) polymorphonuclear leukocytes, (2) invading bacteria, and (3) the plasmin mechanism. Table II summarizes in diagrammatic form the

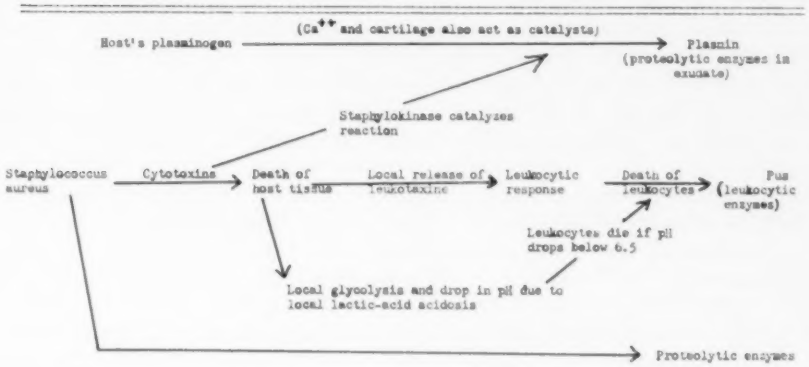
biochemical mechanisms set in action by the invasion of *Staphylococcus aureus*, and indicates the possible sources of the proteolytic enzymes that are found in the pyogenic exudate.

Leukocytic Enzymes

The importance of the proteolytic enzymes produced by the host's own polymorphonuclear leukocytes in the destructive phase of a pyogenic infection in the interspace is difficult to determine. Freyberg stated that pus in the cavity of a joint infected with any type of organism rapidly destroys the articular cartilage as a result of the lytic effect of the digestive ferments found in leukocytes; that

TABLE II

POSSIBLE SOURCES OF PROTEOLYTIC ENZYMES CAPABLE OF DIGESTING COLLAGENOUS INTERVERTEBRAL STRUCTURES



cartilage is digested *in vitro* by proteolytic enzymes in leukocytic autolysates; and that purulent synovial fluids containing upward of 110,000 leukocytes per cubic millimeter are capable of digesting small pieces of cartilage, whereas synovial fluids containing 6,000 to 20,000 polymorphonuclear leukocytes are not.

Compere and Garrison, in reporting their studies on the pathogenesis of osteomyelitis of the spine, described rapid destruction of the cartilage end plates and disc from extension of a hematogenous focus of infection within the body of the vertebra, and attributed this action to proteolytic enzymes formed in the pyogenic exudate. Tuberculous infection, on the other hand, rarely caused complete dissolution of the cartilage plates or the formation of reactive new bone. Martin presented a specimen illustrating the usual tendency toward preservation of the intervertebral space despite the formation of a gibbus and collapse of the body of the vertebra. This is in accord with results of Phemister's experiments of 1924, in which bits of cartilage placed in separate solutions of saline, tuberculous pus, and a suspension of staphylococcus organisms were unaffected, whereas cartilage placed in staphylococcal pus was completely destroyed. Keefer, Holmes, and Meyers also found that leukocytic autolysates from staphylococcal pus digested cartilage. They reported that digestion of the cartilage was not observed in seven samples of synovial fluid from gonococcal arthritis or in three samples from tuberculous arthritis, but in the synovial fluid from two cases of staphylococcal arthritis, there were large numbers of polymorphonuclear leukocytes, and digestion of the cartilage was apparent.

Bacterial Enzymes

Pyogenic exudates contain, in addition to leukocytic enzymes, certain bacterial enzymes capable of hydrolyzing protein. Pincus, studying the etiology of dental caries, demonstrated hydrolysis of collagen by pure cultures of a gram-negative bacillus resembling a strain of *Proteus morgani*. In 1937 Maschmann isolated an enzyme from *Clostridium welchii* that would digest gelatin and collagen, and for it coined the name collagenase.

In elaborating on this work MacLennan, Mandl, and Howes studied the bacterial digestion of undenatured collagen (fresh Achilles tendon of the rabbit) and denatured collagen (azocoll, a commercial preparation of a dried powdered hide mixed with a red azo dye), using various strains of a number of organisms. They were able to show that certain strains of *Staphylococcus aureus*, *Streptococcus pyogenes*, *Bacillus mesentericus*, *Bacillus cereus*, and *Proteus vulgaris* produced enzymes that hydrolyzed the azocoll powder, but not the undenatured collagen. On the other hand, they found eighty-two strains of *Clostridium histolyticum* and twenty strains of *Clostridium welchii* that hydrolyzed undenatured collagen. From this it would appear that the enzymes produced by the staphylococcus itself have no lytic effect on the living collagen of the dog's disc and that perhaps the most important role of the staphylococcus is the production of kinases that activate the host's plasmin mechanism, as suggested by Lack and by Lack and Rogers.

Using the electron microscope, Mandl, MacLennan, and Howes studied the effects of trypsin and collagenase on undenatured collagen. They observed that with collagenase the fibrils became indistinctly tapered and lost their cross striations as if they were being washed away, whereas with trypsin the fibrils were fragmented transversely.

Prior to selecting clostridial collagenase and X-108 as proteolytic enzymes to put in the intervertebral spaces of our dogs, we immersed sections of the distal articular surfaces of rabbit femora in solutions of various enzymes and a phosphate buffer (pH 6.8) for twenty-four hours at a temperature of 36 degrees centigrade. Figures 4-A through 4-D show the effectiveness of clostridial collagenase and X-108 in removing articular cartilage. Other enzymes produced little or no visible change.

Despite the demonstrated complete dissolution of the cartilaginous surfaces of rabbit femora *in vitro* caused by collagenase and X-108 these same enzymes, when placed in the curetted intervertebral spaces of the dogs in these experiments, had no demonstrable effect. Whether this was due to inactivation of the enzymes by circulating antitryptic substances in the living animals or to a species difference has not been determined. Thomas showed that although immature rabbit cartilage is affected by the intravenous injection of crude papain, mature cartilage is not.

Plasmin Mechanism

Plasmin is a protease derived from serum plasminogen, which can be activated by diverse agents, including the kinases of staphylococci and streptococci. Lack and Rogers demonstrated liberation of chondroitin sulphate from the ground substance of bits of cartilage immersed in a buffered solution of plasmin at pH 6.2. They concluded that if sufficient plasminogen is present in the vicinity of cartilage *in vivo* it is possible that activation by the kinases produced by staphylococcus and streptococcus infections may be sufficient to cause considerable loss of chondroitin sulphate from the ground substance, with consequent damage to the mechanical properties of the cartilage.

Lack and Rogers also stated that synovial fluid normally contains a plasmin inhibitor, and that the degree of intra-articular proteolysis depends on the

relative amounts of inhibitor and plasmin. They found that bits of cartilage placed in a suspension of disrupted leukocytes alone were not digested; they also found that leukoprotease is not in itself an important agent in chondrolysis.

From our experience it is evident that a single application of chondrolytic enzyme is not enough to bring about complete dissolution of the disc and cartilage plates. Probably, enzymatic destruction of the intervertebral disc infected by *Staphylococcus aureus* occurred chiefly because an excess of plasmin was sustained by a constant supply of staphylokinase. The belief that the inflammatory reaction leading to intervertebral fusion must be a sustained process is supported also by our clinical impression that in the intervertebral infections treated with antibiotics, intervertebral fusion had not occurred so rapidly or surely as in the infections for which antibiotics were not given. At the present time we are studying a group of dogs that are receiving penicillin for interspace infections induced by a strain of *Staphylococcus aureus* sensitive to this drug. Although our studies are not yet complete, we have been able to prevent fusion of infected segments by administering penicillin.

In some instances the dosage with penicillin was not begun until two weeks after infection. The negative results under these circumstances also suggest that inflammatory reaction of short duration does not provide the proper chemical environment for bone repair, and that a reaction sustained over a period of at least several weeks is necessary to do so.

Whether or not the absence of a synovial membrane and the consequent deficiency of antiplasmin enhance the reaction in the interspace is a matter of conjecture. Burns and Janes were unable to destroy the articular cartilage in the knees of dogs by a single injection of a suspension of live *Staphylococcus aureus*. Although the joints were swollen and hot for forty-eight to seventy-two hours, there was no residual evidence of joint destruction, and cultures of the knees taken two weeks later were sterile.

SUMMARY

Sound bony ankylosis of the spine almost invariably occurs after pyogenic infection in the intervertebral space. Animal experiments were undertaken to study the mechanism of this process as a possible means of inducing ankylosis of the spine artificially.

A single lumbar interspace in each of seventy-four adult mongrel dogs was treated with an irritant (sodium morrhuate, talc, potassium stearate and oleate, or croton oil), a proteolytic enzyme (sterile human gastric juice, clostridial collagenase, or X-108), or a suspension of bacteria (killed *Staphylococcus aureus*, killed *Neisseria gonorrhoeae*, or living *Staphylococcus aureus*).

Solid intercorporeal fusion was obtained in all the twenty-three animals treated with living *Staphylococcus aureus*. Only one fusion was obtained in the fifty-one animals treated in other ways, and in this instance there appeared to have been a surgical infection in the interspace.

A single inflammatory stimulus does not appear to be sufficient to induce interbody fusion in the dog's spinal column. A sustained process in which the intervertebral disc and cartilaginous plates are digested by proteolytic enzymes appears to be required to provide the chemical environment in which the proliferation of new bone and fusion can occur.

NOTE: The authors express their appreciation to Mr. Glenn Christensen for help in preparing specimens, to Dr. F. R. Heilman for preparation of bacterial suspensions, to Dr. G. A. Fleisher for assistance in enzymology, and to Dr. J. H. Grindlay and Dr. J. M. Janes for their helpful suggestions.

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A Study of Strontium^{85} Metabolism in the Normal and in the Castrated Mouse

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One theory maintained that the first cause of osteoporosis on a steroidal basis is old age with its atrophy of bone, and that this atrophy is at least partly attributable to decreased production of anabolic steroids. Testosterone, an androgen produced by the interstitial cells of the testes, is known to promote tissue anabolism and has been assumed to have a direct, stimulating effect on anabolic processes. However, there is some question of whether its action is mediated directly or indirectly on bone by inhibition of the adrenals [1,30]. Treatment with testosterone was reported to be the only procedure which averts the action of estrogen on the bones of the mouse [14]. However, Urist, Budy, and McLean [43] found in their experiments that testosterone, in proportions as high as sixty to one, did not inhibit the effect of α-estradiol benzoate. The mode of action of the anabolic steroids—estrogen and androgen—has been extensively studied in a wide variety of animals since Kyes and Potter reported their results in pigeons. The diversity of the subjects studied and the species peculiarities present in many instances make transfer of obtained values difficult, if not impossible. It is known that estrogen stimulates endosteal-bone formation in birds and mice with production of increased density [15], whereas a deficiency of estrogen and androgen has been observed to result in decreased density of bone in animals and man.

The purpose of this study is to determine in mice the effect of castration on bone. Radioactive isotope evaluation was employed because continuous results can be quantitated by this method. Initially, it was necessary to establish the fact that measurable chemical alteration did occur in mouse bone after castration. Hence, prior to the more extensive isotope studies, a small series of animals was studied by chemical techniques. Detailed observations of the results of these studies follow.

METHODS AND MATERIALS

Male Swiss breeder mice, fed on a standard laboratory diet and kept at normal room temperature, were used in all the experiments performed. The animals were anesthetized by ether, and castration was performed intraperitoneally. The femora were chemically analyzed as follows:

1. Wet-weight determinations were made on scraped, cleaned fresh bone within an hour of removal from the whole animal;

2. Dry-weight determinations were made on the same tissue after extracting for twenty-four hours with low-boiling anhydrous petroleum ether (30 to 60 degrees centigrade, boiling). The extracted bone was dried in a vacuum desiccator for one week or until constant weight;

3. The difference between wet weight and dry weight was designated as *water;*

4. The dry bone was extracted for forty-eight hours with 10 per cent trichloroacetic acid to separate acid-soluble minerals from acid-insoluble protein.

The acid extract was made to volume with additional 10 per cent trichloroacetic acid, and aliquots were used for determination of calcium and phosphorus;

5. Calcium was precipitated from the acid extract with ammonium oxalate after neutralization with ammonium hydroxide (pink to phenolphthalein). The oxalate precipitate was washed once with an aliquot volume of 1 per cent ammonium hydroxide wash. The washed precipitate was converted into calcium oxide by ashing in a muffle furnace for four hours at 600 degrees centigrade. The oxide was dissolved in an aliquot of dilute hydrochloric acid. The acid solution was used for measurement, using the Beckman DU-photomultiplier-oxyacetylene-flame equipment at 443 millimicra wave length. (This procedure should remove all cationic and anionic interferences, in particular those due to sodium, potassium, phosphate, oxalates, and magnesium);

6. Phosphate in the trichloroacetic-acid extracts (Step 4) was measured by the method of Fiske and Subbarow;

7. Residue or matrix was determined by the difference between wet-bone measurements and the sum of $Ca_3(PO_4)_2$ calculated weight plus so-called water;

8. Nitrogen in the trichloroacetic-acid insoluble residue was measured by transferring the entire so-called ghost of the bone remaining after Step 4 to a micro Kjeldahl flask in which digestion and distillation occurred followed by titration. Protein was calculated from the nitrogen values.

To determine the biological turnover of the radioactive isotope in mice, soluble strontium[85] chloride was diluted with 0.9 per cent sodium chloride so that ten microcuries were contained in 0.25 milliliter. The animals studied were injected intraperitoneally with ten microcuries and a similar amount was injected into a serum bottle filled with distilled water, the bottle being of such size as to approximate the volume of each mouse. Immediately after injection the animal was counted in a cardboard box three by three by two inches to determine the exact amount of strontium[85] injected. This amount was then compared with the standard, which was counted in a similar manner. All counting was performed with a heavily shielded scintillation counter at fourteen inches from the source to the crystal. This distance was chosen so that during the radioassays the position of the mouse in its container would have no appreciable effect on counting efficiency. A total of 10,000 counts was collected so that the intrinsic counting error would show a standard deviation of 1 per cent. The total injected dose in each mouse, as calculated by comparison with the standard, was normalized to 100 per cent and the subsequent disappearance of the strontium[85] from the body of the animal was then calculated on this basis. In all subsequent measurements, the animals were compared each day with the counting rate of the standard, thus lessening the chance of error because of variations in counter efficiency or positioning. This method of calculating the total injected dose has been standard in our laboratory and was chosen to eliminate measurement variations in the injected amount of strontium[85], since the amount injected is not critical so long as the total dose in each animal does not deviate appreciably from the standard.

THE EFFECT OF CASTRATION ON SOME OF THE CHEMICAL VALUES OF BONE

Experimental Procedure

Twenty-four male mice, ten weeks of age and from twelve litters, were divided into two groups. One group of twelve animals was castrated while under ether anesthesia; the other group, the litter mates of the animals in the first group, was maintained as a control. A castrated animal and its control were then sacrificed at weekly intervals for a total of nine weeks. The oldest animal sacrificed at the conclusion of this experiment was thus nineteen weeks of age. Three of

TABLE I

RESULTS AFTER ASHING THE FEMORA OF NORMAL MICE AND DETERMINING VALUES BY FLAME PHOTOMETRY

Weeks†	Ash Weight (*Milligrams*)	Calcium (*Milligrams Per cent*)		Phosphorus (*Milligrams Per cent*)		Calcium:Phosphorus Molar Ratios	Residual Fraction (*Per cent*)
1	41.4	11.6	28.0	3.32	5.6	2.71	66.4
2	48.8	15.1	30.9	4.33	6.1	2.70	63.0
3	39.9	10.5	26.3	3.76	6.2	2.17	67.5
4	37.9	10.0	26.4	3.65	6.7	2.12	66.9
5	49.6	12.1	24.4	3.88	6.4	2.41	69.2
6	57.2	14.7	25.7	4.73	7.7	2.41	66.6
7	47.0	9.5	20.2	3.42	6.1	2.16	74.7
8	44.1	12.6	28.6	3.67	6.6	2.67	64.8
9	58.0	14.5	25.0	4.39	6.2	2.56	68.8
Average*	47.1 ±7.0	12.3 ±2.1	26.2 ±2.6	3.91 ±0.47	6.4 ±0.6	2.43 ±0.20	67.4 ±3.3

* With standard deviation.

† Weeks after the opposite, paired animal was castrated (see Table II).

TABLE II

RESULTS AFTER ASHING THE FEMORA OF CASTRATED MICE AND DETERMINING VALUES BY FLAME PHOTOMETRY

Weeks	Ash Weight (*Milligrams*)	Calcium (*Milligrams Per cent*)		Phosphorus (*Milligrams Per cent*)		Calcium:Phosphorus Molar Ratios	Residual Fraction (*Per cent*)
1	48.6	18.3	38.7	5.086	10.48	2.79	50.82
2	41.5	13.46	32.45	4.38	10.56	2.37	56.99
3	54.4	16.2	29.8	5.72	10.5	1.45	59.7
4	49.4	14.0	28.4	5.14	10.4	2.11	61.2
5	61.0	12.3	20.2	5.72	9.4	1.66	70.4
6	49.9	9.75	19.6	5.24	10.5	1.47	69.9
7	44.3	9.05	20.4	4.63	10.4	1.51	69.2
8	65.3	11.75	18.2	6.33	9.8	1.44	72.0
9	46.4	8.25	17.8	4.36	9.4	1.47	72.8
Average*	51.1 ±7.54	12.40 ±3.43	25.0 ±7.4	5.10 ±0.79	10.1 ±0.71	1.58 ±0.54	64.79 ±7.8

* With standard deviation.

the animals died during the period of the experiment and, together with their controls, were discarded from the series.

Results

The percentage of calcium in the femora of non-castrated mice was found to be 26.2, with a standard deviation of ±2.6. The percentage of phosphorus was 6.4 with a standard deviation of ±0.6. The average water content in these femora was 29 per cent. The average calcium to phosphorus ratio in the normal mouse femur was determined to be 2.43±0.2. (These data are shown in full in Table I; similar determinations for castrated animals are shown in Table II.) In Figure 1 the percentage of calcium in the femora of castrated animals is shown as a function of time after castration; the normal value is illustrated showing the average value plus or minus one standard deviation. Whereas the percentage of calcium in the femora of normal mice appeared to remain relatively constant with increasing age (Table I), the calcium values in the castrated animals were mark-

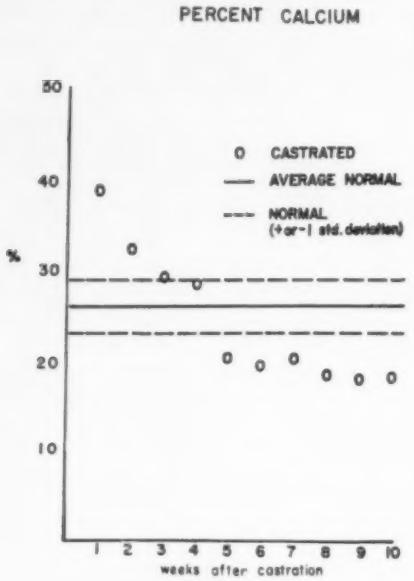

FIG. 1

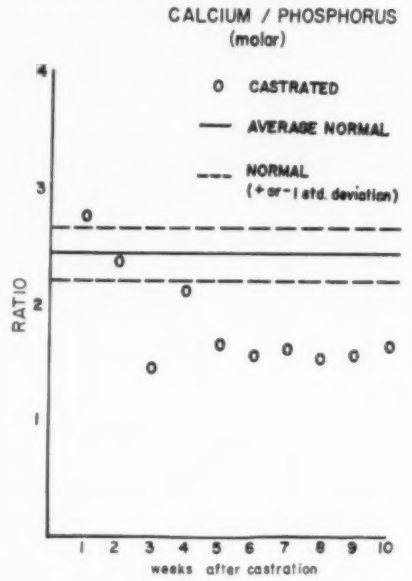

FIG. 2

Fig. 1: The percentage of calcium in the femora of castrated animals is shown as a function of time after castration, together with the average normal value and its standard deviation.

Fig. 2: Calcium-phosphorus ratio in castrates compared with the average value for normal mice.

Fig. 3: Percentage of residue together with the average norm is plotted as a function of time after castration.

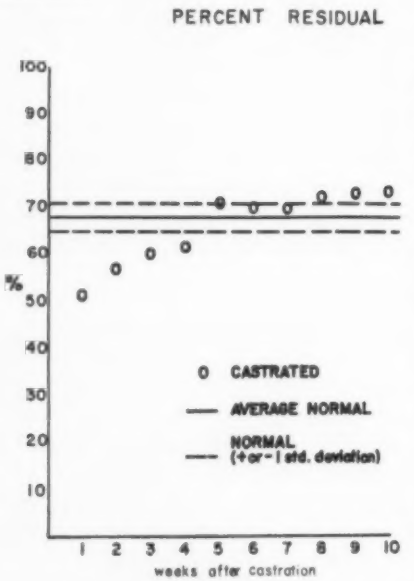

FIG. 3

edly elevated in the period immediately following castration and then fell off progressively with time. After the fifth week these values were found to be uniformly below the normal range and appeared to have reached a level of reasonable stability.

The phosphorus levels in the castrates were all appreciably above the level found in even the highest normal range. No change was found nine weeks after castration. The average value for the normal mouse was 6.4 ± 0.6 per cent and the average value for the castrated mouse was 10.1 ± 0.71 per cent (Tables I and II).

Figure 2 shows the calcium to phosphorus ratio in the castrates compared with the average normal value plus or minus one standard deviation. No significant change in this ratio was found until approximately four weeks after castration; it was then observed that there was an appreciable decrease in the ratio, which progressed until a stable state was reached after the fifth week. Since the phosphorus value was found to be relatively stable during this period, this decrease is almost entirely a reflection of the change in calcium percentage.

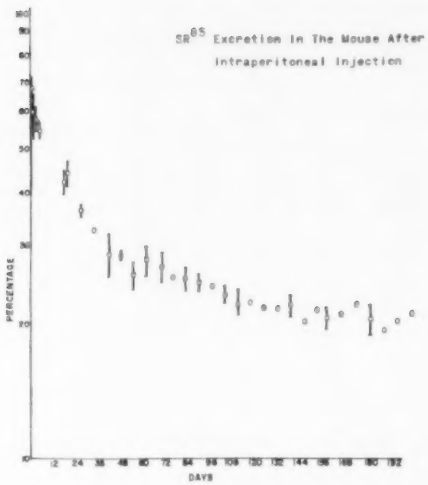

FIG. 4

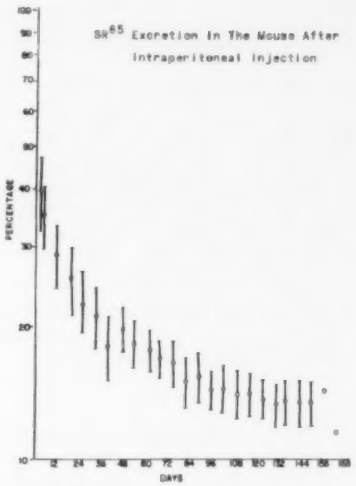

FIG. 5

Fig. 4: The results of whole body assays performed up to 210 days after sacrifice showing percentage of strontium^{85} retained. The vertical lines on each data point, both here and on each figure throughout, refer to plus or minus one standard deviation.

Fig. 5: The results of whole body assays performed up to 171 days after sacrifice showing percentage of strontium^{85} retained. Note the variations in absolute values, as compared with Fig. 4.

In Figure 3 the percentage of residue or the percentage of matrix in the femora of the castrated animals was plotted, together with the average normals, again as a function of time after castration. Although the general trend of the curve for the castrates shows progressive increase, the actual variation beyond the fourth week is comparatively minor. Early measurements, however, show a readily observable decrease to levels definitely lower than the normal range.

It is realized that the number of animals used is too small to attribute any statistical significance to these reported values. What is of primary interest in the study is the established trend, which can be compared with the results of the radioactive determinations.

DETERMINATION OF BIOLOGICAL TURNOVER OF STRONTIUM^{85} IN MICE

Experimental Procedure

To investigate the biological turnover of strontium^{85} in mice, nine animals, eight weeks of age and from two litters, were studied. In a group of four litter mates, one animal died and was discarded from the series. On the remaining three animals *in vivo* whole body assays were performed periodically up to 210 days after intraperitoneal injection of the isotope. The other group of five litter mates was counted periodically up to 171 days after injection of the isotope.

Results

It is important to note that this technique is based on a whole body assay, which measures the total quantity of the retained radioactive isotope in the body, regardless of its distribution. The decrease of retained activity with time indicates not the elimination of strontium^{85} from bone alone, but the actual release of the radioactive isotope from the entire body. The percentage of the administered dose retained by the first group, followed for seven months after injection, is shown in Figure 4 and is typical of our observed strontium^{85} turnover rates and similar to those of other investigators.

TABLE III

DISTRIBUTION OF STRONTIUM^{85} IN MICE FIVE DAYS AFTER TEN MICROCURIES INTRAPERITONEAL INJECTION

	Counts Per Minute Per Gram of Wet Weight					
Mouse No.	Femur 1	Femur 2	Liver	Skin	Spleen	Muscle
1	1.84×10^6	1.97×10^6	5.0×10^2	8.5×10^2	3.3×10^2	6.3×10^2
2	1.60×10^6	1.84×10^6	4.0×10^2	8.0×10^2	2.6×10^2	5.7×10^2
3	2.36×10^6	2.70×10^6	7.0×10^2	9.0×10^2	5.9×10^2	16.0×10^2
4	1.47×10^6	1.30×10^6	4.6×10^2	8.4×10^2	4.7×10^2	3.0×10^2
5	1.44×10^6	2.58×10^6	4.5×10^2	3.9×10^2	3.0×10^2	4.0×10^2
Average*	1.74×10^6	2.08×10^6	5.0×10^2	7.6×10^2	3.9×10^2	7.0×10^2
	$\pm 0.38 \times 10^6$	$\pm 0.57 \times 10^6$	$\pm 0.37 \times 10^2$	$\pm 2.08 \times 10^2$	$\pm 1.38 \times 10^2$	$\pm 5.21 \times 10^2$

* With standard deviation.

TABLE IV

RATIO OF STRONTIUM^{85} IN BONE COMPARED WITH OTHER ORGANS

	Counts Per Minute Per Gram of Wet Weight			
Mouse No.	Liver	Spleen	Skin	Muscle
1	3650:1	5730:1	2250:1	3010:1
2	4350:1	6700:1	2130:1	3010:1
3	3570:1	4320:1	2830:1	1550:1
4	3000:1	2950:1	1655:1	4470:1
5	4500:1	6410:1	5220:1	4800:1
Average*	3814:1	5222:1	2817:1	3368:1
	±614:1	±1570:1	±1400:1	±1309:1

* With standard deviation.

At least two phases may be readily identified in this figure: an initial relatively rapid turnover which lasts for approximately forty-eight days, followed by a slower decrease for the remaining period. A third phase of very slight release is strongly indicated at the end of this time.

Although the second group of five animals shows the same relative turnover rates, the absolute values vary, presumably because of litter differences. The disappearance rate of this group is illustrated in Figure 5.

DISTRIBUTION OF STRONTIUM^{85} IN THE MOUSE

Experimental Procedure

To determine quantitatively that the disappearance of strontium^{85} from the body after initial equilibration was due primarily to mobilization of the isotope from the skeleton it was necessary to know the distribution of strontium^{85} early in this phase. Five litter mates eight weeks of age were injected with strontium^{85}, as just described, and were sacrificed on the fifth day. Liver, spleen, muscle, skin, and femur were weighed wet and then assayed in a standard well scintillation counter.

Result

The distribution of strontium^{85} in various tissues is shown in Table III; the ratio of strontium^{85} deposited in bone to that in the other organs is illustrated in Table IV. The high concentration of strontium^{85} in bone indicates that at five days after injection over 99 per cent of the retained radioactive isotope could be accounted for in the skeleton. The decrease of retained radioactivity subsequent

TABLE V

RATIO OF STRONTIUM^{85} IN BONE TO THE AVERAGE OF SOFT-TISSUE ORGANS IN RELATION TO TIME

Hours after Injection	1	2	4	6	12	24	48	72	120	312	336
Group I	47	89	165								6890
	65	81	191								5745
Group II				173	287	807	1803	2080	3331	7757	
				188	304	1028	1300	2126	2317	6237	
Average*	56	85	178	181	296	918	1552	2103	2824	6997	6317
	±4.0	±5.7	±58.2	±11.2	±12.0	±15.6	±355	±32.5	±715	±1320	±695

* With standard deviation

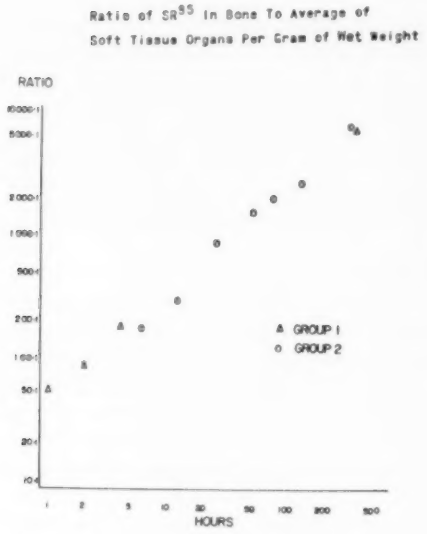

FIG. 6

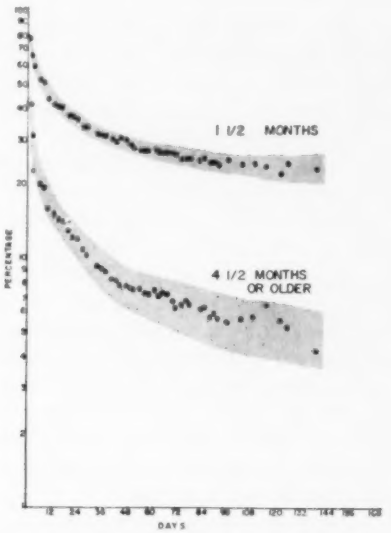

FIG. 7

Fig. 6: *In vitro* measurements of ratio of strontium^{85} in bone to soft tissue.
Fig. 7: The standard deviation of both groups are in the shaded areas. Note the greater error in the older group when litter mates were not utilized.

to this time must, therefore, reflect primarily the release of strontium^{85} from bone.

THE VARIATION WITH TIME OF THE DISTRIBUTION OF STRONTIUM^{85} IN THE MOUSE

Experimental Procedure

To determine the relationship of selective deposition of strontium^{85} to time, eight litter mates six and one-half weeks of age were injected with strontium^{85}, as previously described, and two were sacrificed at one hour, two at two hours, two at four hours, and two at fourteen days after injection. At autopsy, liver, spleen, kidney, stomach, muscle, skin, heart, lungs, testes, intestine, brain, diaphragm, pancreas, thymus, adrenals, and the right and left femora were weighed wet and then counted in a standard well scintillation counter. Fourteen litter mates six and one-half weeks of age were injected; two were sacrificed at six hours, two at twelve hours, two at twenty-four hours, two at forty-eight hours, two at seventy-two hours, two at 120 hours, and two at 312 hours. Organs were

collected, weighed, and assayed in a manner similar to that performed in the first group in this experiment.

Result

The specific activity of strontium^{85} per gram of wet soft tissue was determined for all the organs mentioned. This activity was compared with the activity per gram of wet femora. An over-all ratio of the activity of strontium^{85} in bone to the activity in all the soft-tissue organs was then obtained, using an unweighted average of the activities in these fifteen organs. The ratios for the two groups of mice are tabulated in Table V and the average ratios shown graphically in Figure 6.

EFFECT OF AGE ON RETENTION OF STRONTIUM^{85}

Experimental Procedure

To evaluate the effect of age on retention of strontium^{85} in bone, five litter mates, one and one-half months of age, were injected with strontium^{85} and whole body assays were performed periodically up to 140 days. Five non-litter mates more than four and one-half months of age were similarly injected and assayed periodically up to 140 days.

To evaluate in more detail the effect of age on retention, four litter mates five months of age, five litter mates five and one-half months of age, and four litter mates six and one-half months of age were injected with strontium^{85} and whole body counts done up to 119 days.

Result

The comparison of the retention of strontium^{85} in the group one and one-half months old with that of the group four and one-half months old or older is illustrated graphically in Figure 7. In the group one and one-half months old, the average standard deviation for the forty-one measurements is ± 8.1 per cent, with no point deviating more than 8.6 per cent from the average value up to sixty-two days. Greater standard deviations are observed after sixty-two days because of the lowered counting rate.

A greater variation was observed in the group of mice four and one-half months old and older, with the average standard deviation of this group being ± 19.6 per cent. However, complete separation of the results with no overlap was maintained throughout the entire series.

The magnitude of the standard deviation in the group of non-litter mates (five animals four and one-half months of age and older) demonstrates the necessity for using litter mates for the identification of small differences (Fig. 7). Therefore, for a closer examination of the effect of age on retention, similar measurements on litter mates of five, five and one-half, and six and one-half months of age were obtained (Fig. 8).

It is of interest here to note that whereas the chemical studies showed no significant change in the percentage of calcium, phosphorus, or residue in mice of varying ages, the radioassays showed a significant alteration in strontium retention in animals with an age difference of no more than two weeks.

EVALUATION OF THE EFFECT OF CASTRATION ON RETENTION OF STRONTIUM^{85}

Experimental Procedure

To evaluate the effect of castration on retention of strontium^{85}, thirty-four male Swiss mice five months of age from three litters were utilized. Twenty-two of these were castrated and twelve were kept as controls. The castrates and con-

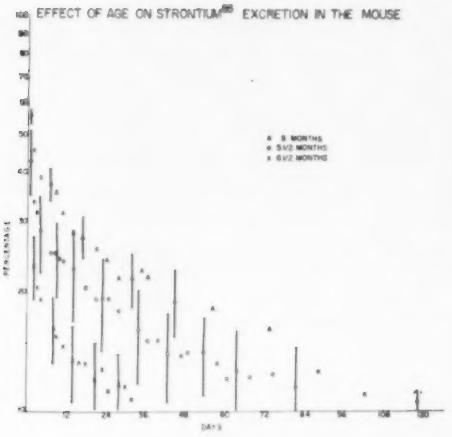

FIG. 8

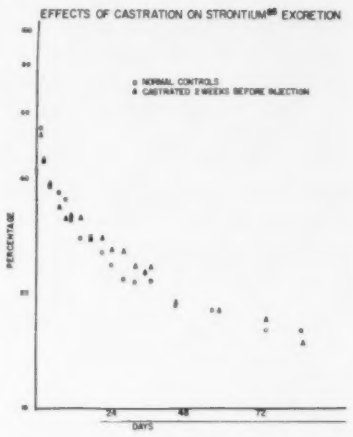

FIG. 9

Fig. 8: Up to the time the animal is six and a half months old, the age of the animal continues to exert a significant influence on the degree of retention. Variation of litter groups may be observed by comparison with Fig. 7. Since the same litter groups have not been maintained in the two figures, the five-month curve in this illustration cannot be compared with the four-and-one-half-month curve of Fig. 7.

Fig. 9: Animals injected two weeks after castration show no significant difference from the normal mice in the retention of strontium^{85}.

trols were divided into three groups. Strontium^{85} was injected into the animals in Group I, composed of twelve castrates and four litter-mate controls, two weeks after castration. In Group II, five castrates and four litter-mate controls, the animals were injected four weeks after castration and in Group III, five castrates and four litter-mate controls, strontium^{85} was injected eight weeks after castration.

Result

In the group injected with strontium^{85} two weeks after castration, there was no observable difference between the retention of strontium^{85} in the castrates and in their litter-mate controls (Fig. 9). However, in Figure 10 definite difference in retention is noted in the group injected four weeks after castration. This difference in the retention of strontium^{85} between castrates and their controls is found to be even greater in the group injected eight weeks after castration (Fig. 11).

DISCUSSION

The mouse has been shown to be an excellent experimental animal for the study of bone metabolism [33,42]. The response of the mouse to castration has been fairly extensively studied, although there is little quantitative evidence of the effect of castration on mouse bone [12]. In the investigation of the possible effects of castration on organs and systems specifically related to bone metabolism, it was demonstrated that castration in the mouse causes a slight decrease in the ratio of kidney weight to body weight and a fall in the percentage of cuboidal cell capsules to approximately the female level for animals of the same age [10]. Kochakian and Stettner demonstrated a decrease in kidney weight of about 38 per cent. Selye noted that large doses of testosterone produced no change in renal blood flow or glomerular filtration rate. The effect of castration on the mouse adrenal is less well known. In general, the influence of gonadectomy is reflected in the portion of the adrenal known as the X zone. This zone has been observed in the adrenal cortex of male mice, especially during the first weeks of life, and

tends to disappear at sexual maturity. After prepubescent castration in the male mouse the X zone persists [11,19]. It has been shown that androgens cause premature disappearance of this zone in immature male mice; these results were confirmed by Starkey and Schmidt and by Martin. However, it has not been determined whether this effect is directly on the adrenal or whether it is mediated through the anterior pituitary. The effect would appear to be direct since it has been shown that testosterone propionate can maintain the adrenal cortex in hypophysectomized-castrated animals [46]. The effects of castration on the accessory sex glands in the mouse suggest that any andromimetic activity in the adrenal of the mouse is concentrated in the X zone. However, it has been noted that the reactions of

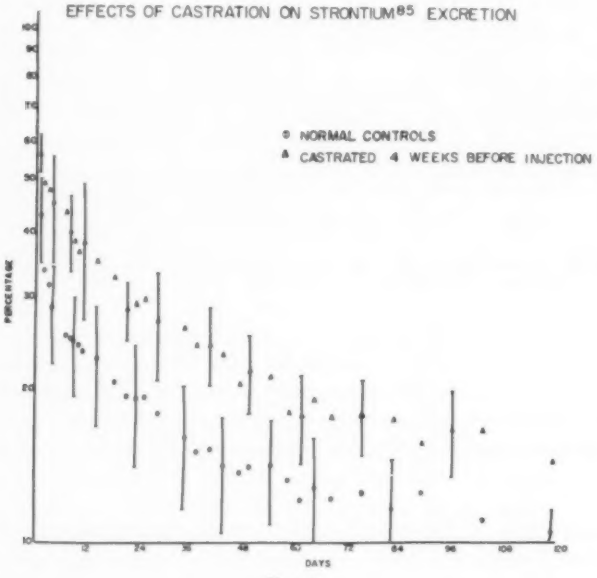

FIG. 10

Animals injected with strontium[85] four weeks after castration.

the accessory sex glands of the mouse are complex, and that the role of the X zone can only be stated tentatively.

It was postulated that the human adrenal produces at least two hormones that affect the formation of bone [2]. Although similar hormones have not been definitely identified in the mouse, certainly in the animals in this study there may have occurred a compensatory increase in the adrenal cortical hormones of the glucocorticoid type. However, this effect was not profound since the calcium loss was noted at a time after castration when compensatory adrenal activity could have been expected anyway. In relation to fluid retention, deSmet noted an initial loss of water in rats of both sexes after castration, but observed that the proportion of water lost decreased progressively thereafter. He found that the initial loss of water and subsequent restoration of equilibrium followed the same general trend as that followed by the change in concentration and distribution of lipids.

Radioactive strontium was employed in this study since it has been demonstrated that there is close similarity between the biological behavior of radioactive calcium and strontium [20,28,31], and it is generally [24,31], although not uniformly [8], assumed that strontium is metabolized by bone in a manner somewhat similar to calcium. The absorption of strontium from the gastro-intestinal tract

varies markedly [24,39] and is affected by the calcium intake before, during, and possibly shortly after the intake of strontium [25,32]. It was demonstrated that parenteral administration of radioactive strontium eliminates to a large degree the effects resulting from differences in absorption of strontium[85] from the gastrointestinal tract [8]. No significant difference was demonstrated in the results obtained when strontium was administered as a chloride, lactate, or gluconate salt [31]. After parenteral administration of radioactive strontium, the isotope is excreted in both the feces and the urine. In mice [31,44], the larger fraction is excreted in the feces.

The retention and excretion of radioactive strontium in the bones of rabbits

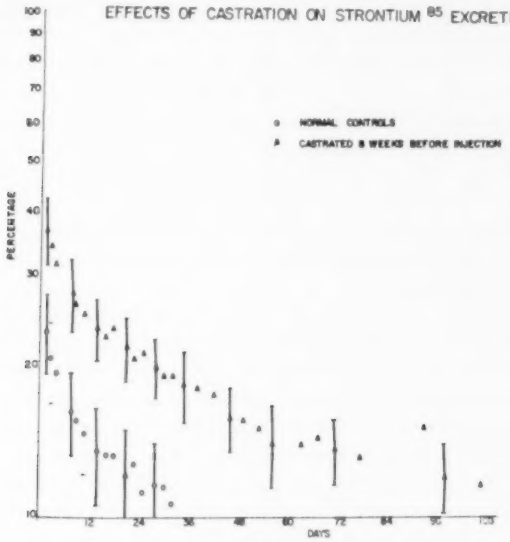

FIG. 11

Animals injected with strontium[85] eight weeks after castration show an additional retention of 20 per cent over the groups in Fig. 10. At no point on the curve was there an observable overlap between the castrates and their controls.

and rats were previously reported in terms of power functions of elapsed time. These values were determined by daily collections of excreta and by serial sacrifice of animals to obtain bone specimens for assay [3,29]. External counting techniques for the determination of the daily loss of activity in the living animal permit variation in the physiological status of the animal and subsequent quantitative evaluation of the effect of this variation on the animal's metabolic status [26].

In the evaluation of *in vivo* distribution of any radioactive material, consideration must be given not only to the sites that concentrate the substance most avidly but also to the excretion or release of the material. The avidity of bone for strontium has been noted by many observers [3,5,6,16,20,34]. Tables III and IV and Figure 6 demonstrate that within twenty-four hours the majority of the retained isotope is deposited in bone. Singer and Armstrong studied large rats, employing radioactive calcium, and determined specific activities of the femoral diaphyses, femoral epiphyses, humeri, lumbar vertebrae, and total skeleton. The values were different on the first day but these differences decreased thereafter until nearly identical values for all the bones were obtained on the fifty-second day. These findings indicate that an intraskeletal redistribution of the exchangeable isotope occurs during this same period.

Since strontium is rapidly deposited in bone, the excretion curves may be evaluated by considering the isotope as being distributed in a two-compartmental system with the skeletal compartment accounting for 99 per cent of the retained material and the kidney and intestine clearing a fractional amount of the isotope which becomes available through equilibration of the isotope in the skeleton with that in the plasma. Four different basic mechanisms may be postulated to explain the variation of the excretion curves of strontium ^{85} between the normal and castrated animal. The difference in absolute amounts excreted may be due to either a difference in bone avidity or a variation in clearance of the isotope by the kidneys or intestinal tract. A variation in the slopes of the excretion curves may in turn be due to either a change in binding of the strontium^{85} to bone or a difference in metabolic activity related to castration.

A comparison of the excretion curves of the normal and castrated animals in Figure 10 indicates that the slopes of the clearance curves are roughly parallel from five to seventy days after administration of the isotope. This indicates that if there is any change in the binding of strontium^{85} or any change in metabolic activity, these changes have little effect on the excretion of the isotope. On the other hand, a significant change in initial distribution of the isotope is readily apparent in the castrated animals. For this reason, unless a large change in total excretion is present after castration, the primary effect of castration must be a variation in initial bone uptake.

As just noted, it has been extremely important to control rigidly the age of the animals studied. It has been observed that the rate of skeletal growth in relation to the timing and duration of a particular study may have a considerable effect on the experimental results. Speckman and Norris, employing rats from four to 428 days old, noted that retention of strontium^{85} varied as much as fourfold between animals of different ages. Comparison of the retention curves of animals one and one-half months old with those of animals four and one-half months of age demonstrates that in the younger animals there is decreased retention of strontium, as would be anticipated. Although the initial uptake may be high in young animals, the rapid rate of remodeling of the growing skeleton permits the strontium taken up by bone to become available for excretion shortly after injection [9,21]. In the mature animals thirty-six days after injection of the isotope, at the mid-point of the experimental period, the retention of the isotope in the animals five months old was approximately 30 per cent less than that in animals five and one-half months old. At the same period of the experiment the retention was decreased an additional 20 per cent in the animals six months old. In other words, a difference in age of as little as 10 per cent resulted in significant differences in excretion (Fig. 8).

A similar dependence on time is shown by the differences in strontium^{85}-excretion curves in normal and castrated animals. The excretion curves of mice injected with strontium^{85} two weeks after castration show no significant deviation from normal. Animals injected four weeks after castration, however, demonstrate a significant increased retention compared with the non-castrated animals. In the comparison of normal and castrated mice injected eight weeks after castration an increase in retention of 30 per cent was found in the castrates at the mid-point period of observation.

In the chemical analysis, no significant variation in bone composition was observed until approximately four weeks after castration. At that time a loss of calcium of from 20 to 40 per cent was observed and this loss continued through nine weeks without significant alteration. The castrated animals injected with strontium^{85} four weeks after castration showed an increased retention of the

isotope of approximately the same percentage. The loss of calcium from bone in the castrated animals might have been anticipated because of the anabolic effect of testosterone. Apparently, in the mouse the anabolic effect of the adrenal steroids is not adequate to compensate for the loss of testosterone. No significance can be attached to the elapsed period of four weeks before the trend of calcium loss from bone becomes established, and certainly some period of latency would be anticipated.

The increased retention of strontium occurring during the period of calcium loss cannot be explained by us on the basis of present data. The existence of osteoid as a prerequisite for mineral deposition has long been known, and considerable emphasis has been placed on the diminution of protein storage as a factor in the decreased density of bone in the senile or the postmenopausal state. Whedon noted that it seems impossible to prove by conventional balance techniques that failure to make matrix is the sole defect. Theoretically, loss of androgen should result in increased excretion of urinary nitrogen with diminished ability of the body to perform anabolic processes. However, the primary mode of entry of labeled inorganic elements into bone is by ionic exchange [4,7,17]. An ion in solution may replace an ion of the same atomic species in the crystal structure of bone or the exchange may occur between ions of different species. Equimolar exchange of strontium ion for calcium ion both *in vivo* and *in vitro* is well established [18,21,41]. It is generally accepted that ions in the plasma in contact with bone surfaces may readily exchange with ions located in the surface of bone crystals. This exchange is considered to be a physical phenomenon which does not result in the accretion of bone. Since the animals utilized in the castration series were young adults, the factor of age and new crystal formation is not significant in the increased degree of retention. Thus, the increased retention of strontium may be primarily the result of the replacement by strontium ions of the calcium ions lost from the crystal lattice of bone after castration.

CONCLUSION

Variation of as little as 10 per cent in the age of animals employed in radioactive strontium studies produces significant alteration in retention of the isotope.

As would be anticipated there is calcium loss from mouse bone after castration.

There is retention of radioactive strontium in castrated mice to a degree comparable with calcium loss. This would suggest the hetero-ionic replacement of calcium ions by strontium ions.

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(Continued on page 1380)

The Treatment of Profundus Division by Free Tendon Graft*

BY R. GUY PULVERTAFT, F.R.C.S., DERBY, ENGLAND

Division of the profundus tendon beyond the insertion of the sublimis may be treated by primary suture when wound conditions permit, with good expectation of success. If primary repair is not advisable, a secondary suture may be considered if division of the profundus has been close to its attachment. Division of the profundus tendon proximal to the sublimis insertion is best treated by skin suture only, with a view to definitive surgery later.

The subject under discussion is the late treatment of profundus division. The choice lies between arthrodesis of the terminal interphalangeal joint, tenodesis, or restoration of tendon action by tendon grafting. The decision depends on several factors: the age of the patient, the condition of the finger and hand generally, and the occupation and wishes of the patient. The operation of tendon grafting is one of some magnitude for what is, after all, a comparatively minor disability. It should not be advised unless the patient is determined to seek perfection and the surgeon is confident of his ability to offer a reasonable expectation of success without risk of doing harm. *Primum non nocere* is a sound principle to remember at all times in reconstructive surgery.

It is my practice to advise tendon grafting for the index and long fingers in the majority of persons because of the important role of the thumb and index and long fingers in pinch. I advise it for the ring and little fingers only when the patient requires this action on account of special interest or occupation, such as a musician or skilled technician. For those employed in general labor and for those who desire a quick and certain result of a less perfect nature, I believe it is better to arthrodese the terminal interphalangeal joint. For children, I consider it is good counsel to advise grafting, as the lengthy recovery period is of no significance and one cannot foretell what may be the child's later requirements.

The replacement of divided flexor tendons by a free tendon graft is one of those essentially personal operations in which only the particular surgeon concerned knows what he can honestly offer to his patient. In 1952 Rank and Wakefield said, "The day has passed when odd successes with flexor tendon repair call for any comment of admiration". We have reached the stage when a patient should be clearly told the prospects of success. We must not forget that a divided tendon does not appear to the patient to present the difficult reconstructive problem that we know it to be. Our advice is more often than not accepted without question, and it is easy to fall into the error of being uncritical of our own results.

The results of tendon repair have been evaluated by two methods:

The first is measurement of the distance by which the finger tip fails to reach the distal palmar crease when the patient makes a fist (Fig. 1). This method of recording results was advocated by Boyes for evaluating the results of tendon grafting to replace divided sublimis and profundus tendons in the digital theca; it has since been widely adopted.

The second method is by noting the range of active movement at the terminal

* Read at the Annual Meeting of the American Society for Surgery of the Hand, Chicago, Illinois, January 22, 1960.

FIG. 1

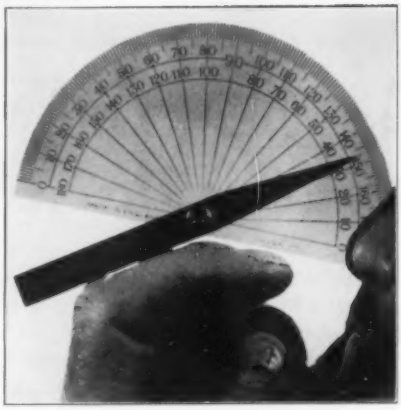

FIG. 2

Fig. 1: Measurement of finger flexion.
Fig. 2: Measurement of the distal interphalangeal-joint range with the finger in semiflexion.

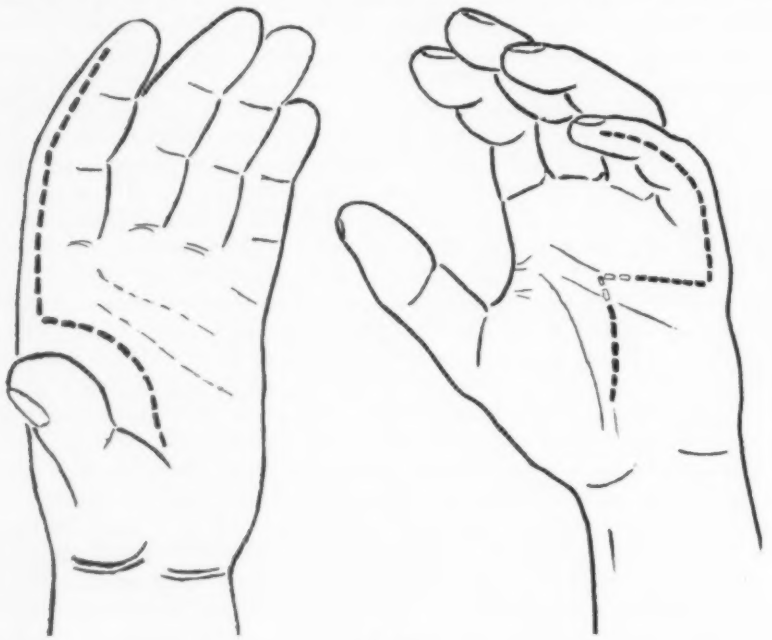

FIG. 3

Incisions used for exposure of the index and little fingers. (Figs. 1, 2, and 3 are reproduced by permission from *The Journal of Bone and Joint Surgery*, **38-B:** 175–194, Feb. 1956.)

interphalangeal joint when the finger is semiflexed (Fig. 2). In this test, the flexion of the distal joint is not assisted by support of the middle phalanx.

Analysis of twenty-two consecutive cases operated on during the years 1942 through 1954 [3] showed that in 86 per cent of cases there was recovery of 30 degrees or more of flexion at the terminal joint. In 50 per cent of the cases there was ability to reach to within half an inch or less of the distal palmar crease (Table I). Extension was complete in all except four of the cases, in which there were flexion contractures of 5, 5, 10, and 20 degrees, respectively. There were no complica-

TABLE I

DATA ON TWENTY-TWO CASES, 1942 THROUGH 1954
(Three Failures Included)

Range of Active Motion at Terminal Interphalangeal Joint when Finger Is Semiflexed		Distance by Which the Finger Tip Fails to Reach the Distal Palmar Crease when the Hand Is in the Fist Position	
Degrees	*Percentage of Cases*	*Inches*	*Percentage of Cases*
30 or more	86	½ or less	50

TABLE II

DATA ON ELEVEN CASES, 1955 THROUGH 1959
(One Failure Included)

Range of Active Motion at Terminal Interphalangeal Joint when Finger Is Semiflexed		Distance by Which the Finger Tip Fails to Reach the Distal Palmar Crease when the Hand Is in the Fist Position	
Degrees	*Percentage of Cases*	*Inches*	*Percentage of Cases*
30 or more	72	½ or less	40

TABLE III

DATA ON TWENTY-NINE CASES, 1942 THROUGH 1959
(Four Failures Excluded)

Range of Active Motion at Terminal Interphalangeal Joint when Finger Is Semiflexed		Distance by Which the Finger Tip Fails to Reach the Distal Palmar Crease when the Hand Is in the Fist Position	
Degrees	*Percentage of Cases*	*Inches*	*Percentage of Cases*
30 or more	92	½ or less	55

tions in these cases, and in no instance was a finger made worse by operation.

In the years 1955 through 1959 twelve cases were treated. One patient, in whom flexor-tendon grafts were placed in the ring and little fingers, could not be traced, but the records nine weeks after operation stated that both fingers came down well and that the result should be very satisfactory in due course. The remaining eleven cases have complete records. Analysis shows a standard of result similar to, but slightly less good than, that in the first series. There was one failure due to rupture of an unusually thin plantaris graft; a tenodesis resulted, which the patient accepted. Including this case, the comparative figures were: recovery of 30 degrees or more of flexion at the terminal joint in 72 per cent, and ability to reach to within half an inch or less of the distal palmar crease in 40 per cent (Table II). Extension was complete in all cases, except for the failure. No finger suffered detriment by operation.

Combining the two groups, a total of thirty-three patients (Table III), and excluding the four failures (three in the first group and one in the second group, all of which were included in the previous analyses), the following figures appear: attainment of 30 degrees or more of movement in 92 per cent and ability of the finger tip to reach to within half an inch or less of the distal crease in 55 per cent.

The operation is similar to grafting for combined sublimis and profundus division. I am very conscious of the fact that the treatment of the divided flexor tendon in the digits has been discussed before this Society on several occasions, and, indeed, at your last meeting in January 1959 there were several valuable and interesting contributions. Nevertheless, it may be of interest to you if I de-

FIG. 4

The plantaris tendon is used as a graft to replace the profundus tendon. The graft in this finger is passed through a transverse tunnel in the distal phalanx. (Reproduced by permission from *The Journal of Bone and Joint Surgery*, **38-B**: 175–194, Feb. 1956.)

scribe the method that I have used for many years, with only minor modifications made in it as I have gained experience. I offer it to you for your criticisms.

At this stage I would like to pay my personal tribute to that unique man, Sterling Bunnell, whose friendship and guidance have meant a very great deal to me. Bunnell visited Great Britain in 1946 and 1952 and made many close friends. He is numbered among the few who have influenced the trend of world surgery and whose influence will be felt for many years to come. As I travel around the world I meet small groups of men doing good work in hand surgery, and in all cases their interest has been aroused by Bunnell, Koch, Mayer, or their immediate pupils. The raised status of hand surgery today is one of the most practical and far-reaching contributions to medicine that your great country has made.

OPERATIVE PROCEDURE

The Graft

The tendon of the palmaris longus, extensor digitorum longus, or plantaris may be used for the graft. My own preference is for the plantaris tendon, which is thin and thus the most rapidly revascularized and at the same time is of sufficient length to provide grafts for two digits, if necessary. It is of equal caliber throughout its length and is easily removed, leaving scars that are virtually invisible. The plantaris is present in approximately 70 per cent of persons. If it is absent, the extensor digitorum longus of the fourth toe is used.

The plantaris tendon is removed through two incisions: one placed to the medial side of the tendo achillis, and the other placed three fingerbreadths posterior to the medial border of the tibia in the upper part of the calf. The tendon lies on the posterior surface of the soleus muscle and is exposed by lifting the medial edge of the gastrocnemius. Paul Brand of Vellore, India, has devised a swift way of removing the tendon by a tendon stripper, using the lower incision only.

The Incisions

The basic principles governing the placement of an incision are that a full exposure be made and that the incision be placed in the immobile parts of the skin. The teaching of plastic surgery is now so widely known that it is inexcusable to employ an incision that may lead to secondary skin contracture.

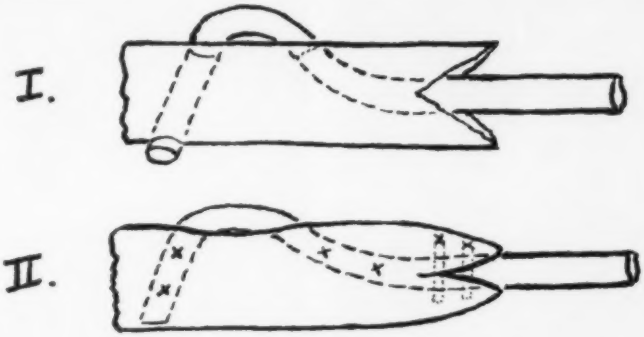

FIG. 5

The interlacing suture used for the proximal junction of a slender graft. (Reproduced by permission from *The Journal of Bone and Joint Surgery*, **38-B**: 175–194, Feb. 1956.)

FIG. 6-A

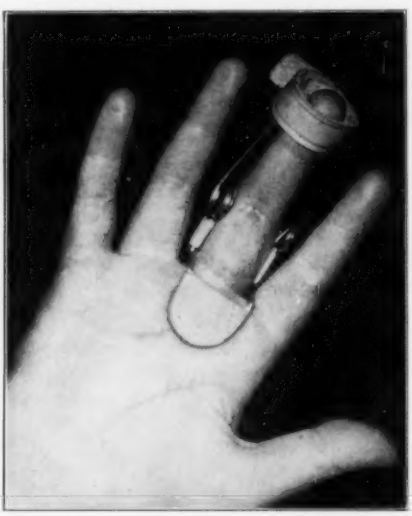

FIG. 6-B

Capener splint used to assist extension.

The index and little fingers are exposed by an exact mid-lateral incision that is continued proximally as far as the respective palmar crease and then extended into the palm along this crease (Fig. 3). In the long and ring fingers, three separate incisions are usually employed: the first is mid-lateral in the finger; the second is transverse, following the distal palmar crease; and the third passes along the proximal part of the thenar crease. When scar tissue, which is unusual in the case of isolated profundus divisions, must be cleared from the base of the digit, the incisions in the finger and the distal palmar crease are joined, and a flap is raised to expose the anterior aspect of the metacarpophalangeal joint. In discuss-

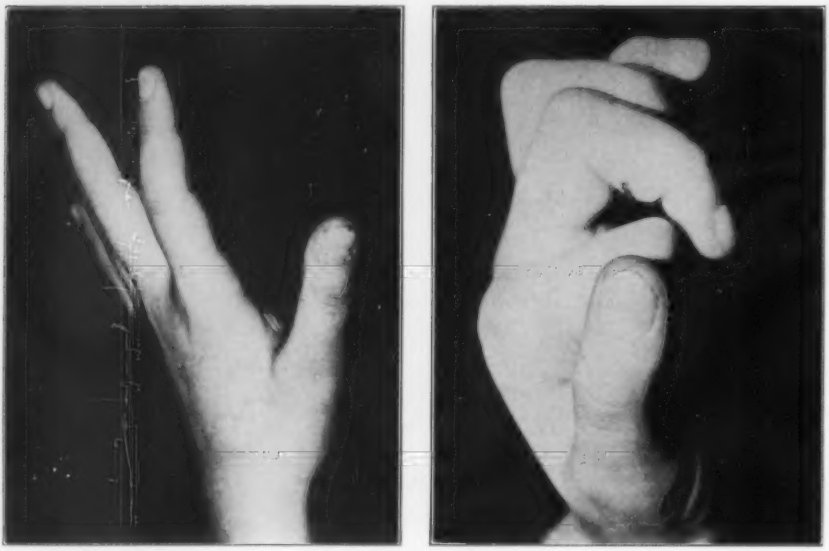

FIG. 7-A FIG. 7-B

Figs. 7-A, 7-B, and 7-C: The result obtained from a toe-extensor graft to replace the profundus tendon of the long finger, divided at the level of the proximal phalanx. The interval between injury and operation was six months.

ing techniques with Tubiana, of Paris, a few weeks ago, he told me that he always exposes the base of the central fingers in this manner. The finger incision is carried straight down to the thecal sheath, leaving the vessels and nerve in the anterior flap. Care is taken to isolate and protect the digital nerve to the radial side of the index finger and to the ulnar side of the little finger, as these nerves cross the operation field. Some surgeons, notably those of the Chicago and Melbourne schools, prefer to raise a superficial flap anterior to the vessels and nerve. This incision would appear to divide the branches of the digital nerve to the volar skin and has never appealed to me. The incision posterior to the nerve offers an exposure fully adequate for a tendon graft.

The Digital Theca

When performing a graft for combined sublimis and profundus divisions, I remove the theca and synovial sheath completely, with the exception of those parts that are left to act as pulleys; this is unnecessary in most cases of isolated profundus divisions, unless the case is a difficult one, with extensive adherence between the divided profundus and the intact sublimis.

The Sublimis Tendon

I have heard the opinion expressed that the uninjured sublimis should be removed, on the grounds that this gives better expectation of a freely sliding graft. I am convinced that this policy is unsound, for none of us has yet been able to achieve 100 per cent success after performing a graft. Removal of an uninjured sublimis tendon cannot fail to be a potential source of harm. The presence of the sublimis tendon, provided it is undamaged, does not prejudice the action of a profundus graft. The graft is inserted along the exact course of the profundus tendon after this tendon has been completely removed (Fig. 4).

FIG. 7-C

FIG. 8-A

Figs. 8-A, 8-B, and 8-C: Result obtained from a plantaris graft to replace the profundus tendon of the ring finger, divided at the level of the proximal interphalangeal joint. The interval between injury and operation was six months.

FIG. 8-B

FIG. 8-C

The Suture

We are all agreed that the suture material should be fine and of such a nature that it causes the least possible tissue reaction. Those who have seen the foreign-body granulomata that form around catgut and thick silk wonder why these materials are ever used in the delicate surgery of the hand. Mukherjee and Douglas studied the relative reactions set up in nerve tissue by nylon, terylene, human hair, silk, and stainless-steel wire and demonstrated clearly that wire is the only ma-

FIG. 9-A

FIG. 9-B

Figs. 9-A, 9-B, and 9-C: Result obtained from a plantaris graft to replace the profundus tendon of the long finger, divided at the level of the proximal interphalangeal joint. The interval between injury and operation was one year.

FIG. 9-C

terial that causes little or no reaction. It has been found in practice that there is little to choose between fine silk (No. 0000 to 000000) and stainless-steel wire, making the choice largely one of personal preference. It has always been my custom to use single-strand wire—40 British wire gauge for the main suture and 43 British wire gauge (No. 000000) for auxiliary stitches. Kinking must be avoided, and the wire is tied by a reef knot, the ends being cut off flush with the knot. The twisted and braided wire is, of course, more supple and a little easier to handle. I have tried both these forms but quickly returned to single-strand wire, on account of both its neatness and the security of the knot.

I find it easier to perform the proximal suture first and later adjust the ten-

sion at the distal suture, but this again is a matter of personal choice, and many surgeons do it the other way around. There is universal agreement that the final tension should cause the digit to take up a position of slightly more flexion than it would naturally assume under anesthesia. The graft does not shrink, and the muscle belly will often stretch out a little to resume its normal length with use.

The proximal suture of a small tendon graft, such as the plantaris, to the larger profundus tendon, is best accomplished by an interlacing suture that is secondarily converted into an end-to-end junction by a fish-tail enclosure of the graft by the end of the motor tendon (Fig. 5). The junction of the graft and motor tendon is then covered by stitching the lumbricalis muscle around it. The distal suture of the graft is made with a Bunnell withdrawal stitch, making sure that the graft end is put snugly into the angle between the bone and profundus attachment. I have found it helpful here to use hollow injection needles, which are passed through the tip of the finger to emerge at the precise point of attachment. The wire suture in the graft is cut from its needle, and its two ends are passed through the hollow needles. The needles are then removed, leaving wires in place ready to tie over a small bolster of wool. When the operation has been completed, the table is tilted feet down and laterally toward the opposite side, and the arm is raised off the table before release of the tourniquet. After a wait of four to five minutes in this position, it is found that bleeding has virtually ceased. It is exceptional for any vessel ligation to be needed. The wound is not closed until full hemostasis has been secured. Drainage is never used. The limb is kept elevated for forty-eight hours after operation.

The Aftercare

The finger is held in company with the normal fingers in 75 per cent flexion for three weeks by means of a steel-wool pack in the palm, crepe bandage, and strapping. The steel wool—a household pan scraper—was shown to me by Erik Moberg, and I have now used it for several years. It has the advantage of providing a splendid compression dressing and does not clog as does wool. When steel wool is used, air can permeate it, and the skin of the hand remains dry and in good condition. At the end of three weeks, this dressing is removed, and a single elastic adhesive strap, one inch wide, is applied to the dorsal surface of the finger for another week. This adhesive strap spans the palm and is attached to the front of the forearm. This strap permits some movement of the finger but prevents excessive extension stress on the graft. The terminal withdrawal stitch is removed at four weeks, and free movements are encouraged. It is, I believe, important to splint the digit in flexion rather than to flex the wrist and allow the finger to be straight. It is easier to overcome adhesions in flexion than to improve the flexion range. Spring splintage (Figs. 6-A and 6-B) can be used to improve extension, but only the patient's voluntary movement can stretch adhesions limiting flexion. A course of treatment in the Physical Therapy Department then follows removal of the withdrawal stitch. All patients are given the Bunnell wood block against which the individual joints are exercised. Supervision is continued until the maximum recovery has been attained (Figs. 7-A through 9-C).

CONCLUSIONS

Restoration of the flexor digitorum profundus in the presence of a normal sublimis by a free tendon graft is justifiable in selected cases.

The sublimis tendon must not be removed.

As the disability caused by lack of profundus action is comparatively slight and can be treated in other ways, tendon replacement should only be undertaken by surgeons of experience.

(Mr. Pulvertaft's references are on page 1380)

Adduction Injury of the Knee with Rupture of the Common Peroneal Nerve

Report of a Case

BY MAX M. NOVICH, M.D., NEWARK, NEW JERSEY

The first report of a traction injury of the external popliteal (peroneal) nerve associated with a closed injury of the knee was made by Platt in 1928. He noted fracture of the fibular styloid process with displacement of bone fragments of varying sizes by the avulsed biceps tendon and rupture of the external lateral ligament.

Watson-Jones in 1931 reported two more cases of this type and described the severe lateral (adduction) strain of the knee with subluxation of the joint which produces this type of injury. He noted that the analogous injury to the elbow involves subluxation of the joint with displacement of the internal condyle into the joint and subsequent palsy of the ulnar nerve.

Platt reported five additional cases in 1940. He described the extensive rupture of the ligamentous and capsular structures on the lateral aspect of the knee joint and the traction injury to the external popliteal nerve (with or without anatomical division) which is caused by adduction of the leg on the thigh.

Highet and Holmes added eight more cases and an excellent review of the neuropathological and neurosurgical features. Involvement of the lateral collateral ligament was noted, although fracture of the fibular styloid was not present in these cases. This comparatively rare lesion is produced, usually, by industrial or traffic accidents; two of the reported cases resulted from football injuries. Smillie called this injury the ligamentous peroneal nerve syndrome.

Sudden powerful adduction of the leg on the thigh by an external force may produce extensive rupture of the capsular and ligamentous structures on the lateral aspect of the knee joint, thus stretching or tearing the common peroneal nerve (Figs. 1 through 4).

The pathological changes in the nerve extend far beyond the site of obvious damage in the form of nerve-fiber degeneration, intraneural fibrosis, and vascular abnormality. Even if macroscopic continuity of the nerve is preserved, paralysis may result because of damage to the axis cylinders within the nerve trunk. In either case, the prognosis depends on the extent of damage to the axons in the proximal part of the nerve and on the length of time which has elapsed between injury and repair.

In addition to the nerve lesion, there are rupture of the lateral capsule close to the tibial margin, avulsion of the biceps tendon, and tear or avulsion of the external lateral ligament, carrying with it a portion of the fibular styloid.

As noted by Smillie, there may also be associated rupture or avulsion of the iliotibial tract, the popliteus muscle, and the cruciate ligaments. The lesion should be suspected in any adduction-type injury of the knee with signs of injury to the lateral ligamentous and capsular structures. The diagnosis of this syndrome is confirmed if there is a sensory deficit on the outer aspect of the leg and dorsum of the foot and weakness or paralysis of the muscles innervated by the peroneal nerve. If the patient is seen immediately after injury, before the onset of muscle spasm, the obvious instability of the knee might focus attention on the knee and

cause the signs of peripheral-nerve injury to be unnoticed. Subsequently, muscle spasm, anxiety, and excitement may also obscure the severity of the knee injury, in which case examination of the knee under anesthesia might be indicated. If the possibility of peroneal-nerve injury is kept in mind, the diagnosis can be established readily. Faradic and galvanic stimulation can be used if there is any doubt about the presence of a peroneal-nerve lesion.

O'Donoghue called attention to the necessity for early surgical treatment of knee injuries if a major ligament has been torn or avulsed from its attachment.

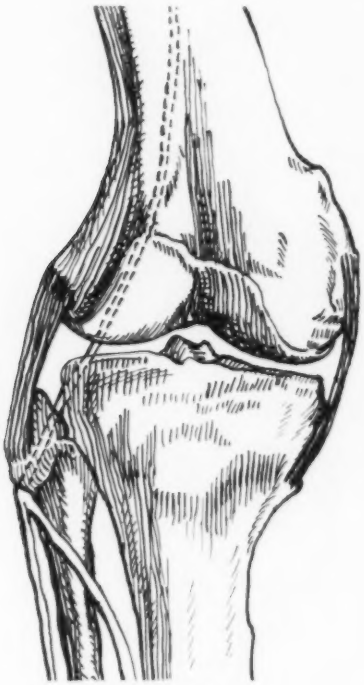

FIG. 1

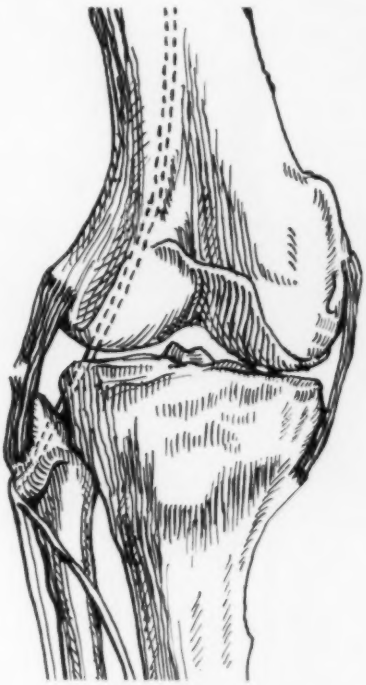

FIG. 2

Fig. 1: Anterior view of the normal knee with the medial and lateral collateral ligaments and the common peroneal nerve with its bifurcation.

Fig. 2: Minor sprain of the lateral collateral ligament; the common peroneal nerve is intact.

Early exploration of the nerve is important; definitive repair should be made as soon as feasible and optimally between the first and third week after injury.

A healthy male, sixteen years of age, was brought to the emergency room of the Beth Israel Hospital, Newark, New Jersey, on September 13, 1957, thirty minutes after sustaining an injury to his left knee in a football game when he was blocked by an opposing lineman. As he was hit, his left knee buckled outward with an audible snap, and he fell backward. He noted immediate pulling pain and the rapid development of swelling, but, initially, he was able to flex and extend the knee.

When first examined there was obvious swelling about the knee, with ecchymosis apparent on the anterior aspect of the leg. Tenderness was found over the lateral aspect of the knee, extending from ten centimeters above to ten centimeters below the joint line. Motion of the knee was painful but flexion and extension were possible through a fairly good range. No mention was made in the record of any abnormality of the foot or ankle, probably because the emphasis, at that time, was on the serious injury to the knee. Roentgenograms of the knee showed no fracture or dislocation.

The patient was admitted to the hospital with a diagnosis of severe contusion and sprain of

the left knee with possible tear of the lateral meniscus. After admission the leg was elevated and ice packs were applied. The following day a foot-drop with numbness on the dorsum of the foot was discovered, and the presence of an injury to the peroneal nerve was recognized and confirmed by a neurological consultant. In the presence of increasing edema and ecchymosis about the knee, a diagnosis of contusion of the left common peroneal nerve was made. The foot was supported by a footboard, and four days after injury thirty cubic centimeters of blood was aspirated from the left knee, after which twenty-five milligrams of hydrocortisone and 150 turbidity-reducing units of hyaluronidase were injected. Marked hypermobility of the knee in adduction was now demonstrated, indicating rupture of the lateral collateral ligament.

Ten days after injury, the physical-therapy department reported that faradic and galvanic stimulation elicited no contractions of the peroneus longus and brevis or anterior tibial muscles;

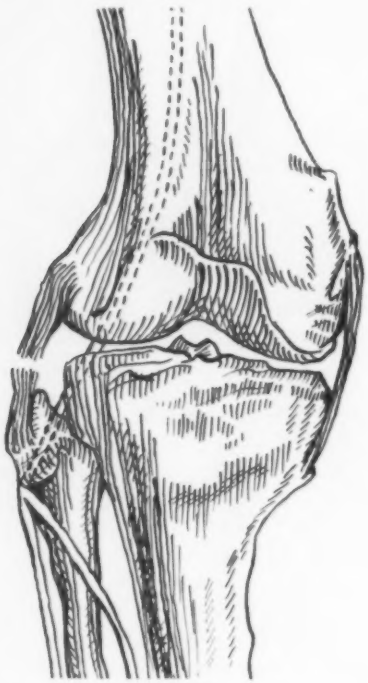

FIG. 3

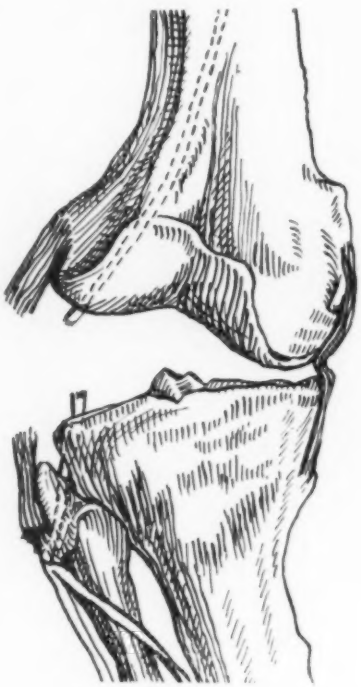

FIG. 4

Fig. 3: Major sprain of the lateral collateral ligament; the common peroneal nerve is intact.
Fig. 4: Major sprain of the lateral collateral ligament; the common peroneal nerve is ruptured.

there were trace contractions of the extensor digitorum longus and brevis muscles. The contractions of the toe extensor muscles suggested that a few nerve fibers remained intact, but complete disruption of the peroneal nerve was found at exploration. The gastrocnemius and toe flexors were normal to manual testing.

On September 25, 1957, twelve days after injury, arthrotomy with exploration of the peroneal nerve was performed, through a lateral approach, by the author in company with a neurosurgeon. The biceps tendon was found to be avulsed from the head of the fibula, the lateral collateral ligament was ruptured, and the lateral joint capsule was torn at its attachment to the tibia. The lateral meniscus was intact, remaining attached to the tibia, and the cruciate ligaments were not ruptured. The peroneal nerve was disrupted at the level of the fibular head, where its distal segment was identified. This was thickened and fibrotic for a distance of approximately two centimeters. The frayed end of the proximal segment of the nerve had retracted to just above the popliteal space.

The torn joint capsule and biceps tendon were repaired by suture. Repair of the nerve was not attempted because it was believed that the extent of the nerve damage in this avulsion injury could not be determined at this time, and because it was thought best to defer repair until the surrounding hemorrhage and edema had subsided so that tissue reaction in the vicinity of the neurorrhaphy

would be minimized. The nerve ends were sutured together in a side-to-side fashion to prevent further retraction and to facilitate the identification of the nerve at a later date. At the completion of the procedure, the knee was immobilized in a toe-to-groin cast in 15 degrees of flexion. After three weeks the cast was removed. At this time the wound had healed well, and the sutures were removed.

On October 17, 1957, five weeks after injury, the nerve was re-explored under spinal anesthesia. The nerve ends had pulled apart, and the end of the proximal segment was found in dense scar tissue in the center of the popliteal space. The distal segment was identified as a fibromatous mass below the level of the knee joint. The nerve was mobilized proximally and distally, and serial segments were resected until normal nerve fasciculi could be identified. A rather extensive amount of the proximal segment had to be removed so that the final gap between the nerve ends measured ten centimeters. By mobilizing the nerve further proximally and flexing the knee to 90 degrees, nerve suture with 000000 silk and an atraumatic needle was accomplished, but there was slight tension on the suture line. Although this tension was considered undesirable from a technical standpoint, it was believed that the neurorrhaphy was the best that could be accomplished in this patient. At the conclusion of the procedure a toe-to-groin plaster cast was applied, with the knee in 90 degrees of flexion.

Histological examination of the resected nerve ends revealed intraneural and perineural fibrosis with chronic inflammation. In the sections from the distal segment there were degenerating

FIG. 5

Range of motion and distribution of analgesia at last examination, three years after injury.

nerve bundles with increased amounts of collagenous fibrous tissue surrounding them. In the sections from the proximal segments, degenerating nerve trunks and myelinated nerve bundles were identified, surrounded by extensive cellular and collagenous fibrous tissue. Evidence of nerve-cell regeneration was seen in one area. Small foci of chronic inflammation and tiny foreign-body granulomata were also observed.

The patient's postoperative course was uneventful, and he was discharged from the hospital walking on crutches on November 8, 1957. Approximately six weeks following repair of the nerve, gradual controlled extension of the knee was begun by splitting the cast at the knee and using adhesive-tape straps to permit gradual extension over a period of three more weeks. A drop-foot brace was prescribed.

Orthopaedic re-examination on May 18, 1959, showed excellent range of knee motion with no return of motor power in the anterior tibial, peroneal, or toe extensor muscles. An area of hypalgesia was noted on the lateral distal third of the leg with an area of analgesia on the dorsum of the foot in the region of the first and second metatarsals. These findings were confirmed by the neurosurgeon who re-examined the patient on May 21, 1959, and also found a moderately positive Tinel sign at the upper lateral border of the popliteal space and a slightly positive Tinel sign at the head of the fibula and on the dorsum of the foot. He concluded that the patient showed evidence of regeneration of the sensory portion of the peroneal nerve, but no recovery of function of the motor component. In view of the eighteen months that had elapsed since the neurorrhaphy, he concluded that the prognosis for motor function was practically nil. On June 26, 1959, the left foot was stabilized by a Lambrinudi procedure.

When last examined on October 10, 1960, the patient had no complaints referable to the left knee or ankle; there was numbness on the dorsum of the foot. The knee was stable and had full range of motion. There was slight equinovarus of the front part of the foot but the talus remained

in the position where it had been placed at operation. There was no Tinel's sign in the vicinity of the knee or neurorrhaphy scar. There was no return of motor function but there was evidence of almost complete regeneration of the sensory portion of the superficial peroneal nerve. Analgesia was still present on the dorsum of the foot in the distribution of the deep peroneal nerve (Fig. 5).

DISCUSSION

Requests for information about the absolute and relative incidence of lesions of the lateral collateral ligament of the knee with involvement of the peroneal nerve were sent to orthopaedic surgeons with wide experience in the treatment of athletic injuries. Twenty-nine replies were received; twenty-four physicians had not seen this type of injury, and five reported a total of ten cases.

Almost all the physicians who replied commented on the extreme rarity of this condition among athletes and the population in general. Although ligamentous and other injuries to the knee occur very frequently, apparently the peroneal nerve usually is not traumatized beyond its limit of tolerance.

SUMMARY

A case is reported of a young male athlete with an injury to the lateral collateral ligament of the knee which also involved the common peroneal nerve.

It appears that this injury occurs very rarely, and therefore attention is directed to the clinical signs and symptoms which facilitate diagnosis of this condition.

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Congenital Malformation of the Atlanto-Axial Joint with Dislocation*

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Chronic dislocation of the atlanto-axial joint secondary to congenital anomalies of this region is unusual. The purpose of this paper is to report a case of a hypoplastic odontoid process with forward displacement of the atlas.

ANATOMY

A unique and peculiar anatomical arrangement enables man to nod his head and rotate his neck. The two occipito-atlantal joints allow for backward and forward tilting of the head, whereas the primary function of the atlanto-axial joints is rotation. The atlas, which characteristically has neither a body nor a spinous process, is a bone ring supporting the head on its two lateral masses and surrounding the odontoid process of the axis in a pivotal fashion [4].

The odontoid process articulates with the posterior surface of the anterior portion of the atlas ring and is held in place primarily by the transverse ligament of the atlas and also by the alar and apical check ligaments that bind the odontoid process to the occiput.

Rotation of the atlas about the odontoid process is facilitated by the shape of the contiguous lateral articular surfaces of the first two cervical vertebrae which are nearly circular and flat and lie roughly in the horizontal plane. In contrast to the articulations between the other cervical vertebrae, these lateral joints of the atlanto-axial segment are more anteriorly situated. The spinal cord is suspended from each side of the dura mater by the denticulate ligaments. The spinal dura mater is, in turn, attached to the margins of the foramen magnum, to the second and third cervical vertebrae, and also to the posterior longitudinal ligament. As a result, the spinal cord is relatively fixed in close relationship with the back of the odontoid process and with the anterior aspect of the posterior arch of the atlas.

DISLOCATION

Dislocation of the atlanto-axial joint is not unusual in association with fractures of the anterior or posterior arch of the atlas with or without concomitant fracture through the base of the odontoid process and in association with solitary fractures of the odontoid process [1,10]. Spontaneous dislocation of the atlas may occur as a result of infection, particularly in tuberculosis where there is decalcification of bone and consequent disruption of the attachments of the transverse ligament. With congenital absence of the odontoid process [2,3,6,7,8,11] there is excessive mobility of the atlas on the axis, and with hypoplasia of the odontoid process the check ligaments may fail to control the normal pivotal motion. A rudimentary odontoid process may slide under the transverse ligament, which is probably what occurred in the patient in this report. Subsequently, with chronic dislocation, relaxation or degeneration of the other ligamentous attachments may

* The opinions expressed herein are those of the author and are not to be construed as reflecting official Air Force policy.

FIG. 1

FIG. 2

Fig. 1: Preoperative roentgenogram showing anterior dislocation of the first cervical vertebra on the axis.

Fig. 2: Roentgenogram made through the open mouth, revealing the hypoplastic odontoid process.

occur with further anterior progression of the head and the atlas on the axis.

During the early years of life these congenital anomalies are usually innocuous; however, excessive mobility of the spinal segment may cause repeated minimum injuries to the cervical spinal cord that may lead to late neurological manifestations [5,9]. An additional mild trauma or senescent changes frequently elicit symptoms of cord compression in later life.

REPORT OF A CASE

J.M.N., a white man, twenty-seven years old, an Air Force aircraft mechanic, was hospitalized in August 1956 with the chief complaint of pain in the neck and shoulders. The spontaneous onset of pain occurred twelve months prior to admission and was aggravated by lifting or straining with the upper extremities. There were associated occipital headaches that became persistent. In addition, the patient had recurrent stiffness of the neck, intermittent numbness of both arms, transient weakness of the right upper extremity, and paresthesiae of the hands. The antecedent medical history revealed no trauma or infectious disease involving the neck. The patient was undergoing medical treatment for a duodenal ulcer.

Physical examination revealed normal mobility of the neck, except for 25 degrees of limitation of extension. Localized pain in the neck was produced by flexion of the cervical spine and shrugging of the shoulders. The spinous process of the second cervical vertebra was abnormally prominent. Hyperesthesia was present in the posterior region of the neck and occiput. Deep tendon reflexes were hyperactive symmetrically in all the extremities. There was generalized loss of muscle tone in the upper extremities without localized muscle weakness. Roentgenograms of the cervical spine showed a malformed and shortened odontoid process with forward dislocation of the occiput and atlas (Figs. 1 and 2). The displacement was exaggerated on forward flexion of the neck.

On September 17, 1956, a spine fusion was performed from the occiput to the fifth cervical vertebra using autogenous iliac bone. The procedure was performed under endotracheal general anesthesia with the cervical spine stabilized by Crutchfield-tong head traction and the patient in the prone position, his head supported on a cerebellar head rest.

Examination at the time of operation disclosed partial absence of the laminae and a bifid axis. There was lack of development of the normal ligaments overlying the dura in this region. In order to ensure adequate stabilization, a long fusion was done from the superior nuchal line of the occiput down to the laminae of the fifth cervical vertebra. The occipital bone above the foramen

FIG. 3-A

FIG. 3-B

Postoperative roentgenograms of the cervical spine in the lateral projection with the neck in flexion and extension to demonstrate the integrity of the bone-fusion area extending from the fifth cervical vertebra. The graft is well consolidated and shows no defect eleven months after operation.

magnum and on both sides of the medial nuchal crest was scarified with an osteotome, and thin flaps of the outer bone table were lifted. The spinous processes and laminae of the third, fourth, and fifth cervical vertebrae were denuded to prepare the graft bed. Two large iliac-bone struts were applied to each side of the mid-line, and the proximal portions of the molded grafts were inserted into the prepared occipital bone shelves. The graft was well fixed and it was unnecessary to employ metallic internal fixation. Additional slivers and cancellous-bone chips from the posterior portion of the right ilium were applied to the fusion area. Care was taken to see that no bone pressed directly on the exposed dura. The wound was closed in layers. The postoperative period was uneventful, and the wound healed well. The patient was kept on a Stryker frame with skull-tong traction for three weeks. A Minerva jacket was then applied, and the patient was allowed to walk for the ensuing four months. Five months after operation he was discharged to military duty in a cervical four-poster brace.

The patient worked as a mechanic without difficulty while wearing the neck brace. There was significant regression of the preoperative symptoms. He wore the neck brace for four months and then wore a Thomas collar for three more months. Roentgenograms of the cervical spine made in August 1957 showed a solid fusion (Figs. 3-A and 3-B). Examination at this time revealed the anticipated restriction of nodding and rotation movements caused by the fusion. Thirteen months after surgery the patient was involved in an automobile accident and sustained a severe flexion-extension injury. He was hospitalized elsewhere, and roentgenograms showed no defect of the grafted area and no evidence of other spine injury. He had a sore neck for five weeks that was treated in a Thomas collar. His pain gradually subsided, and when last seen in April 1959 the patient was asymptomatic and working regularly.

SUMMARY AND CONCLUSION

A patient is reported who had progressive neurological changes secondary to congenital malformation and subluxation of the atlanto-axial joint. An occipito-cervical fusion was performed as a prophylactic measure. A severe neck injury of the flexion-extension type thirteen months after operation resulted in only minor symptoms. Had this patient's atlanto-axial joint not been stabilized by a solid fusion prior to this accident, transection of the cord might well have been the outcome.

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Growth Following Transepiphyseal Bone Grafts

An Experimental Study to Explain Continued Growth Following Certain Fusion Operations*†

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In recent years there appeared in the orthopaedic literature a number of studies [2,6,15] describing continued growth in length of apparently solidly fused spines of young children. Other authors [5,9,14] also noted bending of apparently solid fusion masses after operations for scoliosis. Smith and Yu noted continued growth in the capital femoral epiphyses of children after solid extra-articular hip fusions and Hatt described knees in which fusions with long transarticular cortical grafts across the epiphyseal lines did not interfere with growth of the epiphyseal cartilage, despite lack of evidence of breakdown of the graft. None of these observations seemed to us to be consistent with the generally accepted theory that there is no such thing as intrinsic growth or intrinsic bending of fully formed bone. Therefore, it was decided to reinvestigate this problem from both the experimental and the clinical viewpoints. This article deals with the experimental aspects; another article will report our clinical observations.

Previous experimental studies reported in the literature concerning this problem have often been confusing or contradictory. Macewen, after resecting the distal radial epiphyseal cartilage in dogs, found that in one animal the distal radius and the distal ulnar epiphysis had fused to each other and that a diaphyseal synostosis had developed further proximally between the radius and ulna, while the distal ulnar epiphyseal line remained intact. There was no growth at the distal ulnar epiphyseal line. In another dog, the distal end of the radius fused to the ulnar epiphysis without the formation of synostosis elsewhere. In this limb growth of the distal ulnar epiphyseal plate continued exerting distal traction on the radius to the extent that the proximal radio-ulnar joint was subluxated as the result of distal displacement of the radial head. A solid bone bridge, spanning the epiphyseal line, seemed to have stopped growth in the first dog, whereas in the other the ligaments and interosseous membrane were not strong enough to do this.

Friedenberg resected half of the epiphyseal cartilage of the lateral condyle of the femur of six-week-old rabbits and found in some instances ". . . evidence that some growth had occurred in the lateral condyle subsequent to surgery, despite microscopic and roentgenographic evidence of a thin layer of bone fusing the epiphysis to the metaphysis". Animals with a thicker plate of bone showed no evidence of growth. Siffert drilled holes across the distal femoral epiphyses of rabbits and noted "localized central epiphysiodeses" which ". . . slowed growth but did not cause gross epiphyseal deformity because concentric growth was uninterrupted in the unimpaired peripheral portion of the epiphyseal plate".

* Read at the Annual Meeting of the Orthopaedic Research Society, Chicago, Illinois, January 24, 1959.

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Ford and Key, in a similar experiment, noted: "The epiphyseal cartilage away from the hole in its center was usually normal in appearance, but the epiphysis was fused by bone around the periphery of the drill hole". None of these authors attempted to explain how growth continued despite these local epiphyseodeses.

Although a preliminary report of this investigation[8] was presented prior to that of Campbell, Grisolia, and Zanconato, their superb study of trauma to the epiphyseal plate provided an excellent review of experimental work in this field and demonstrated experiments in dogs similar to those reported here in rabbits. They placed large homogenous cortical-bone grafts across the epiphyseal plates and found that these grafts arrested growth of the plates in all of their eight animals.

In clinical orthopaedics the Phemister type of epiphyseodesis is usually successful in suppressing growth; this seems incompatible with some of the findings just mentioned. However, although this operation is essentially a bone graft across part of an epiphyseal line, it is usually performed with two large grafts and with curettement of the remainder of the epiphyseal cartilage. Fractures through epiphyseal lines, pins traversing the growth cartilage, and infections in the epiphyseal area undoubtedly excite bone proliferation within the injured areas and frequently bone bridges of greater or lesser degree must form between the epiphysis and metaphysis. In most cases, however, it seems that the epiphyseal growth force is able to resist partial or complete cessation of growth in these areas. On the other hand, if the bone bridge spanning the growth center is large enough or the growth force of the rest of the cartilage plate is sufficiently disrupted, growth arrest generally results.

It was the purpose of this investigation to seek some explanation for these varied clinical and experimental phenomena that would satisfy the observed facts and be consistent with the basic concepts of bone physiology. Therefore, attempts were made to simulate in experimental animals some of the unexplained clinical problems just mentioned.

METHODS

The obvious and ideal method for the controlled study of spine fusions in young human beings would be spine fusions in young animals. Accordingly, Hibbs and Albee fusions and cortical grafts with screw fixation were carried out in puppies, kittens, and young rabbits, but almost invariably gross pseudarthroses developed at each interspace. Vertebral-body fusions were also tried, but these were associated with a high mortality in these young animals; it was also difficult to fuse a long segment and gross pseudarthroses still resulted in many instances. Our findings agreed with the conclusions of Odelberg-Johnson that gross pseudarthroses form, and that continued growth in length of the spines of young animals routinely occurs after spine fusion. This did not, however, help in explaining what happens to young human spines after apparently solid fusions. It was apparent that the spines of small laboratory animals could not be properly immobilized, and that motion was probably the main factor causing the defects in the graft.

A method was sought by which the complicating factor of motion could be completely eliminated, and the so-called stretch effect of growth alone could be studied. The most simple and direct method that we could devise to eliminate motion consisted in the placement of intramedullary bone grafts across the distal femoral epiphyseal plates in young rabbits. We reasoned that the cancellous bone on either side of the epiphyseal plate would readily fuse to the graft. Then the graft, as a solid strut of bone attached to two solid plates of bone proximal and

distal to the plate would either suppress growth of the epiphyseal cartilage or, if growth continued, the graft or its bone attachments would elongate in some manner. Serial microscopic sections were made at different times after operation because it was believed that gross observations and measurements alone could show only that growth did or did not occur, without indicating how and why it happened.

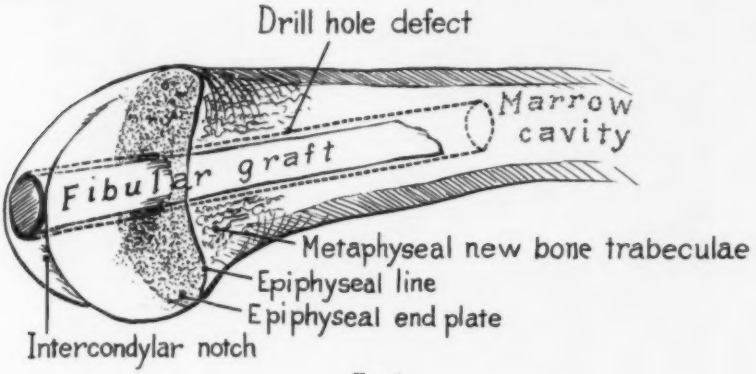

FIG. 1

Method of grafting across the epiphyseal line.

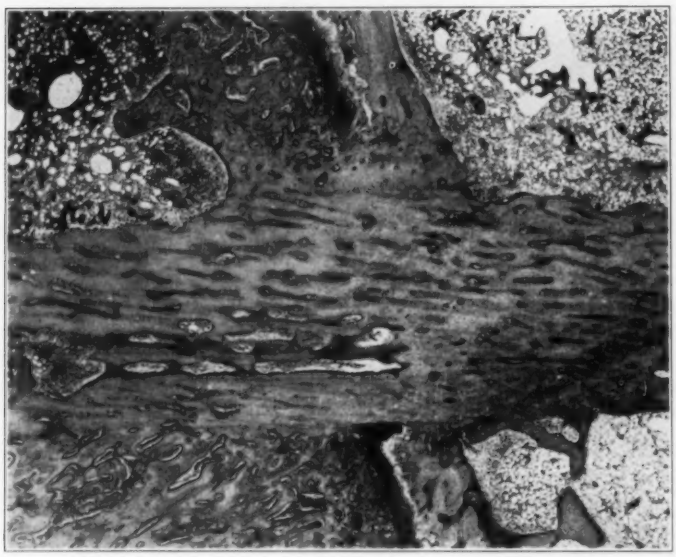

FIG. 2

Fibular graft, twenty-five days after implantation, shows solid bone fusion linking the epiphysis and metaphysis. Growth of the epiphyseal line is completely suppressed. Clamped in a vise of solid bone, the epiphyseal plate is narrow and its normal histological pattern severely disorganized (× 20).

New Zealand rabbits, five to seven weeks old, were used throughout. The animals were anesthetized with nembutal and ether and after surgical preparation both knee joints were entered through lateral parapatellar incisions and the patellae were dislocated medially. Holes, seven-sixty-fourths of an inch in diameter, were drilled on both sides from the intercondylar notch through the

FIG. 3

Site of drill hole only, twenty-five days after operation. There is obvious loss of bone continuity in the defect in the plate, which is filled with fibrous tissue. This tissue shows evidence of stretching, with linear arrangement of the collagen fibers. Growth of the epiphyseal plate is not suppressed (× 20).

TABLE I

MICROSCOPIC FINDINGS IN SEVENTY RABBIT FEMORA

	Fibular Grafts (*Days after Implantation*)				Drill Hole Only (*Days after Operation*)		
	5 to 11	12 to 20	21 to 28	over 28	5 to 11	12 to 20	over 20
Fusion		2	2½*	2		1	½*
Equivocal fusion	6	3	1	1	1		
Equivocal loss of bone continuity	4	5	3	4	9	4	8
Loss of bone continuity			3½*	1		5	3½*

* One side of the epiphyseal line was fused and the other showed continuing growth. Except in these cases, growth was equal throughout the plate all the way out to the periphery.

center of the epiphysis and epiphyseal line and on through the metaphysis into the marrow cavity (Fig. 1). Approximately one-ninth of the area of the epiphyseal cartilage was destroyed by the drill. On the right side, the major portion of the fibula (about three-quarters of an inch long) was removed through a continuation of the same incision. This bone fitted snugly in the seven-sixty-fourth inch drill hole and was pushed gently in until the end was flush with the articular cartilage (Fig. 1). The left side was used as a control and the drill holes were left open. Silk threads or carbon markers placed one centimeter apart on the graft were used in some of the animals, until it was discovered that the microscopic findings were sufficiently informative without them.

The animals were sacrificed at varying times (Table I), the lengths of the femora measured, and then the lower end of each femur decalcified, fixed in celloidin, sectioned, and stained with hematoxylin and eosin. Approximately twenty sections were made from each bone. These were cut in a longitudinal

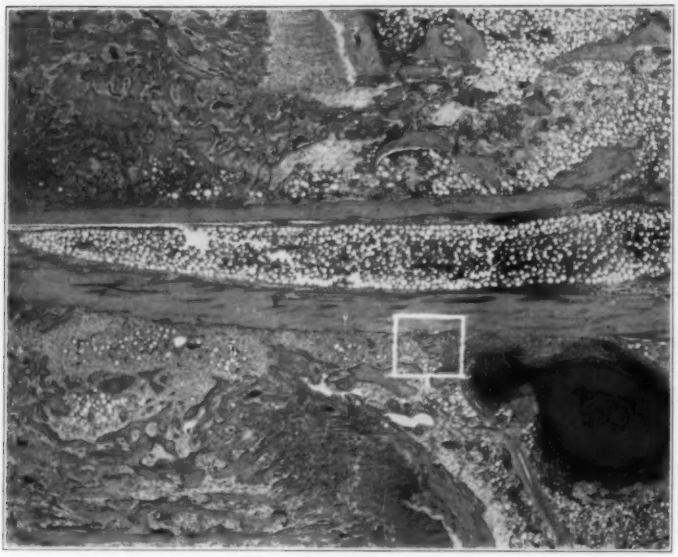

FIG. 4-A

Fibular graft, five days after implantation. The graft is intact; new bone is developing along the margins of the graft and growing across the defect in the epiphyseal plate (above and below graft). There is no solid bone bridge as yet, and the epiphyseal cartilage shows little evidence of suppression of growth (× 20).

FIG. 4-B

Higher magnification (× 200) of area in the box outlined in Fig. 4-A. There is marked cellularity and trabeculae of new bone are attached to the graft.

plane 160 micra apart at the periphery of the femur and 80 micra apart near the center, at the site of the graft. All sections were ten to twelve micra thick.

RESULTS

Thirty-eight femora with drill holes and fibular grafts were sectioned. Ten

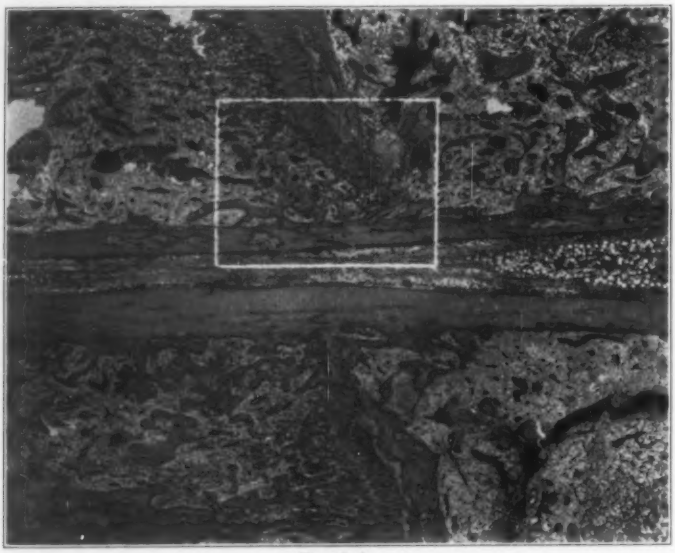

FIG. 5-A

Fibular graft, nine days after implantation. New bone can now be seen attached to the graft in both the metaphyseal and the epiphyseal sides of the plate; new bone has also grown through the defect in the plate. Both the graft and the new bone in the defect are intact and the epiphyseal plate shows evidence of growth suppression. However, the bone graft is being resorbed, particularly in the area enclosed in the box (× 20).

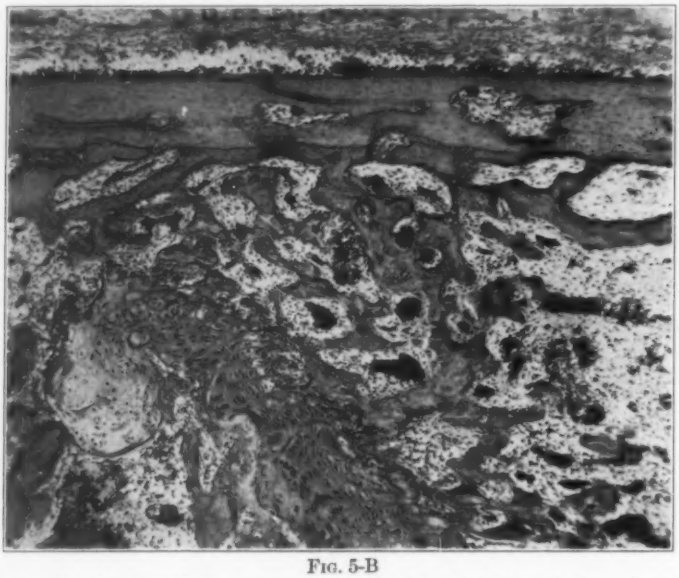

FIG. 5-B

Higher magnification (× 65) of area in the box outlined in Fig. 5-A. Marked new-bone formation and resorption of the bone graft can be seen. Note thinness and irregularity of the epiphyseal cartilage, indicating partial suppression of growth.

were done from five to eleven days after operation, ten from twelve to twenty days, ten from twenty-one to twenty-eight days, and eight from twenty-eight to forty-nine days. Thirty-two control femora with drill holes only (no grafts) were similarly sectioned. Ten were done from five to eleven days after operation, ten

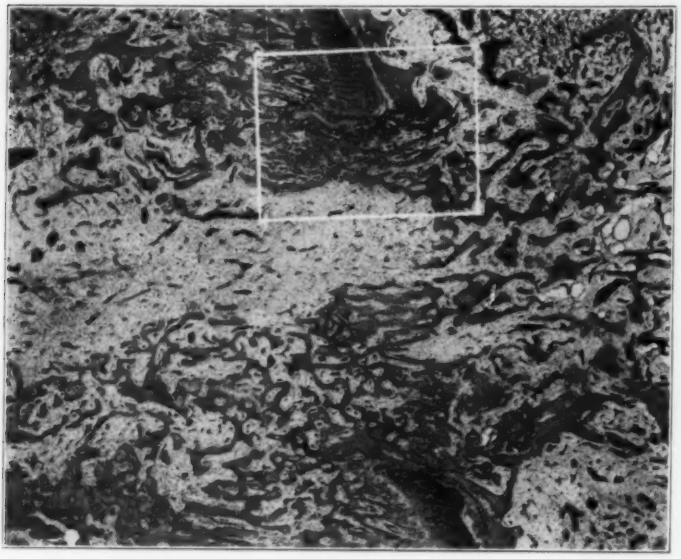

FIG. 6-A

Site of drill hole (no fibular graft) ten days after operation. Well formed new bone, comparable to the cancellous bone seen elsewhere, is apparent in the defect seen in the epiphyseal plate. The quantity of this new bone appears sufficient to block further growth, but the cartilage plate is of normal thickness, with regular columns of cartilage cells. Close inspection of the bone trabeculae within the defect in the plate reveals a zone of increased cellularity extending across the defect parallel to the epiphyseal plate (× 20).

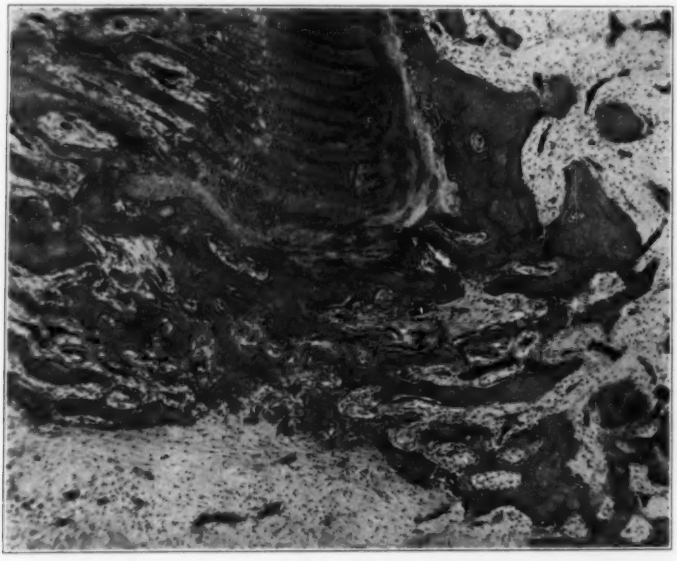

FIG. 6-B

Higher magnification (× 65) of the area in the box outlined in Fig. 6-A shows more clearly the marked cellularity, as well as actual defects in several trabeculae. Although a solid bridge of bone still extends from the epiphysis to the metaphysis, the trabeculae forming this bridge are thin, with many cells around them, suggesting that these trabeculae could be absorbed in short order.

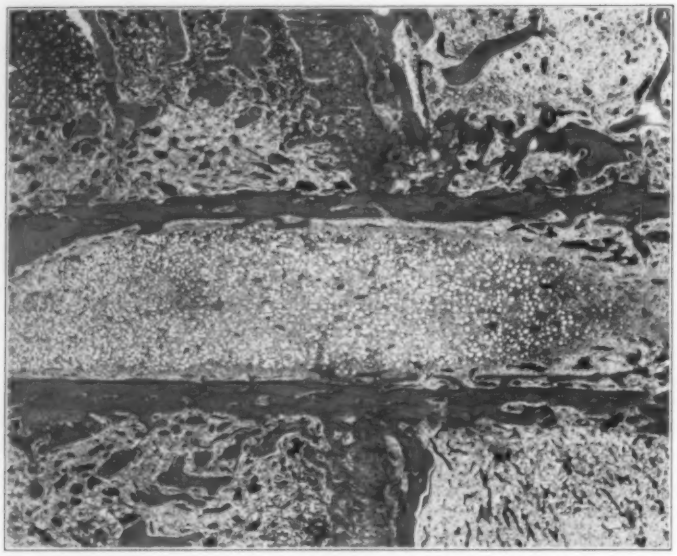

FIG. 7

Fibular graft fourteen days after implantation. Considerable osteoclastic resorption of the graft has now occurred and defects in the graft can be seen in several places. On both the metaphyseal and the epiphyseal sides of the plate new bone has fused to the graft and is replacing the bone in the graft. Although here the epiphyseal plate appears to be compressed, it is not possible to determine at this stage whether the graft and new bone will suppress growth of the epiphyseal cartilage or whether the force of growth and osteoclasis of the graft will cause loss of continuity in the graft, liberating the epiphysis to resume growth (× 20).

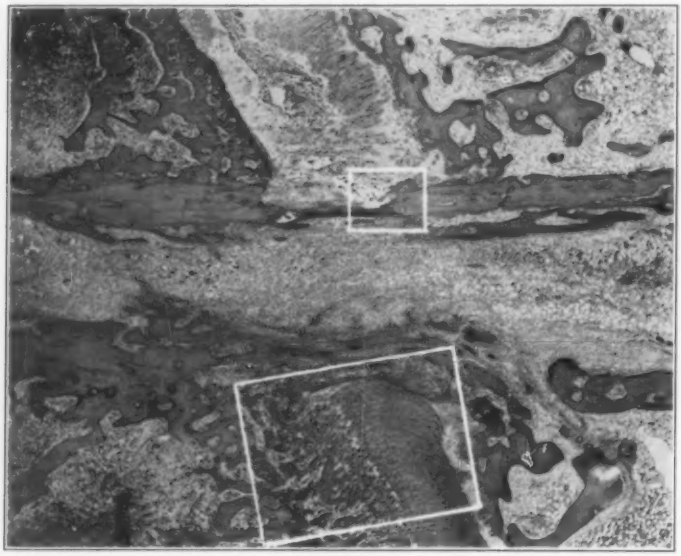

FIG. 8-A

Fibular graft, fifteen days after implantation. The graft has been completely resorbed in the region of the defect in the epiphyseal plate and a clear-cut defect can be seen in the graft. On the lower side of the defect in the plate, new bone can be seen extending from the metaphysis to the epiphysis. Note, however, the very cellular reaction here (× 20).

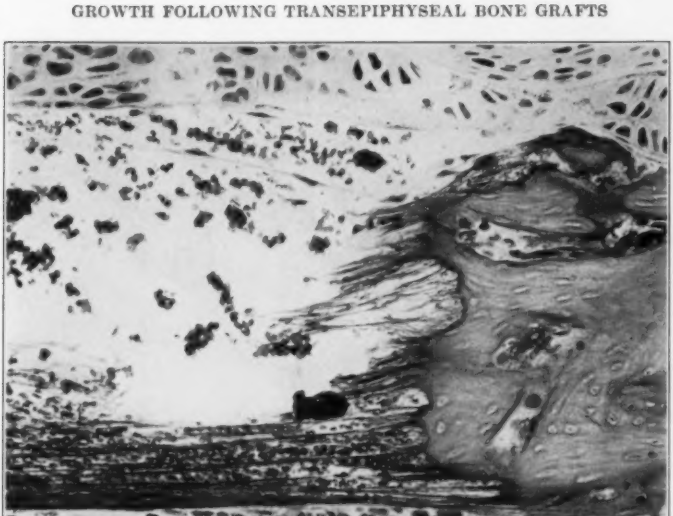

FIG. 8-B

Higher magnification (× 200) of the area enclosed within the smaller, upper box outlined in Fig. 8-A. The bone of the graft on the right appears to be dead. Resorption of this dead graft bone seems to be taking place by solution of the mineral elements while a fibrous network remains behind. This differential absorption seems to be contrary to the present concept that when bone resorption occurs, there is complete resorption of all elements. A similar reaction to dead bone grafts was described by Lacroix [10].

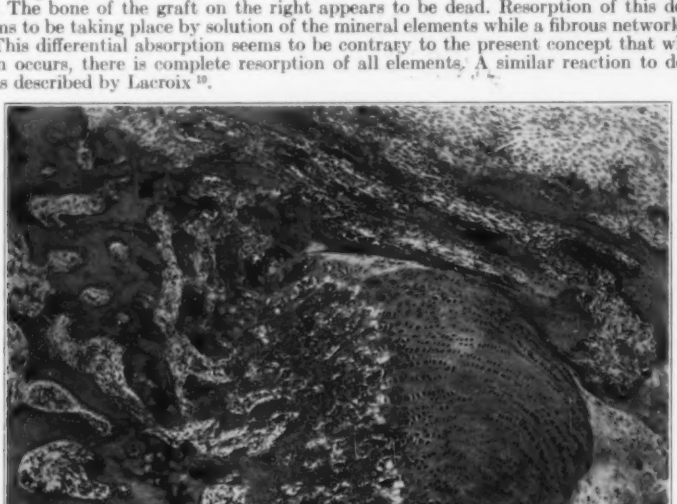

FIG. 8-C

Higher magnification (× 50) of the area enclosed within the larger, lower box outlined in Fig. 8-A. Remnants of cartilage plate in the form of fragments of calcified cartilage (to the left) appear to be separated from the actively growing epiphyseal plate (to the right) by a zone of new metaphyseal bone trabeculae. This finding was interpreted by Lacroix [11] as indicating that growth had temporarily ceased at an earlier stage, with the formation of still-visible, irregular, calcified cartilage remnants. This growth suddenly began again with the restoration of the normal histological pattern of the epiphyseal plate. This sequence of events is comparable to that which occurs after removal of epiphyseal staples. It is presumed that the findings here indicate that when the graft and new bone were intact, growth of the plate was arrested; however, when resorption of the graft caused loss of continuity, growth was allowed to resume.

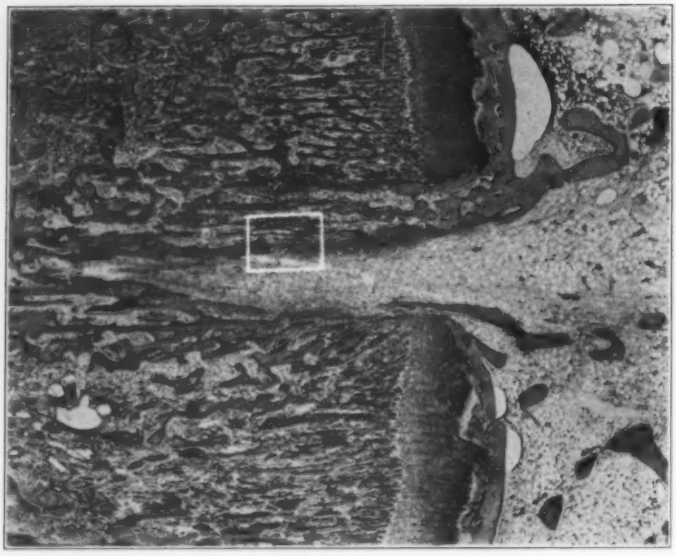

FIG. 9-A

Site of drill hole (no fibular graft), twenty-one days after operation. Although a solid bridge of bone appears to extend from the epiphysis to the metaphysis, the epiphyseal plate appears to be normal, with no evidence of compression. In the metaphyseal region, particularly in the region enclosed within the outlined box, there is a band of increased cellularity extending across the bridge of bone. Within this band, the trabeculae are thin and stretched (× 20).

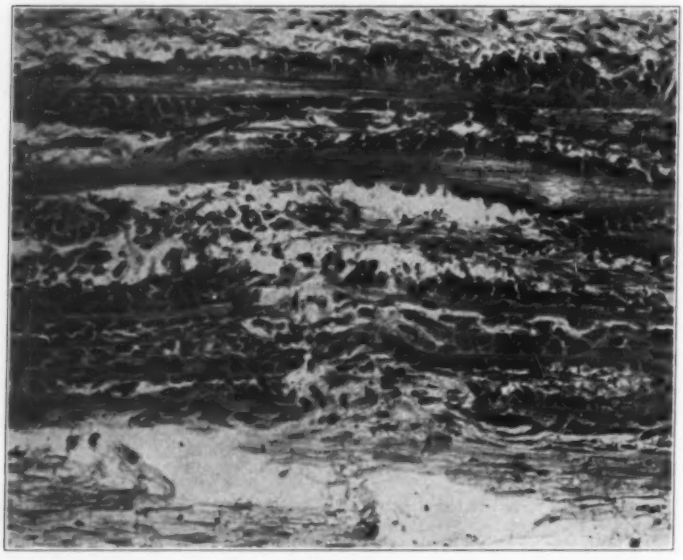

FIG. 9-B

Higher magnification (× 200) of the area enclosed within the box outlined in Fig. 9-A. Note the marked cellularity and the absence of bone. Only numerous bundles of fibers remain. All aligned in the direction of the stretch. Some of these bundles show loss of continuity.

FIG. 10-A

Fibular graft, twenty-eight days after implantation. Despite a massive bridge of bone, which appears to extend from the metaphysis to the epiphysis, growth of the epiphysis seems unimpeded. Here again, there is a zone of increased cellularity (area outlined) deep in the metaphyseal region. This zone extends across the bridge of bone, and, even at the magnification, breaks in the continuity of the trabeculae can be seen throughout (× 20).

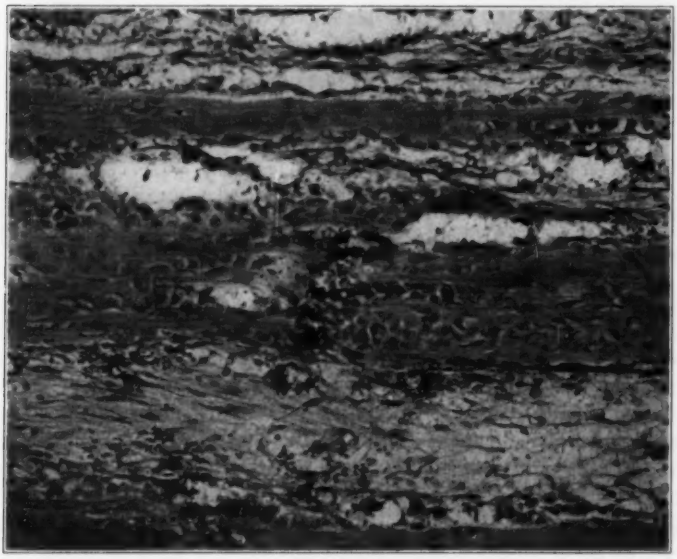

FIG. 10-B

Higher magnification (× 200) of the area enclosed within the box outlined in Fig. 10-A. Note the seemingly stretched fibers and the many cells. In the center there appears to be an actual break in one of the fibers.

from twelve to twenty days, and twelve from twenty-one to forty-nine days. The microscopic findings could be classified roughly into four major categories:

1. Fusion: a solid bridge of bone, extending through the drill hole and joining the epiphysis and metaphysis, and microscopic evidence of marked suppression of growth of the cartilage of the epiphyseal line (Fig. 2);

2. Equivocal fusion: considerable bone extending through the drill hole with no evidence of loss of bone continuity and moderate suppression of growth of the epiphyseal cartilage (Figs. 4-A, 4-B, 5-A, 5-B, and 7);

3. Equivocal loss of bone continuity: considerable bone extending through the drill hole, but definite microscopic evidence of stretch-alignment of the trabeculae, a break in continuity of the bone, and mild to moderate suppression of growth of the epiphyseal cartilage (Figs. 6-A, 6-B, 7, 8-A through 11-B).

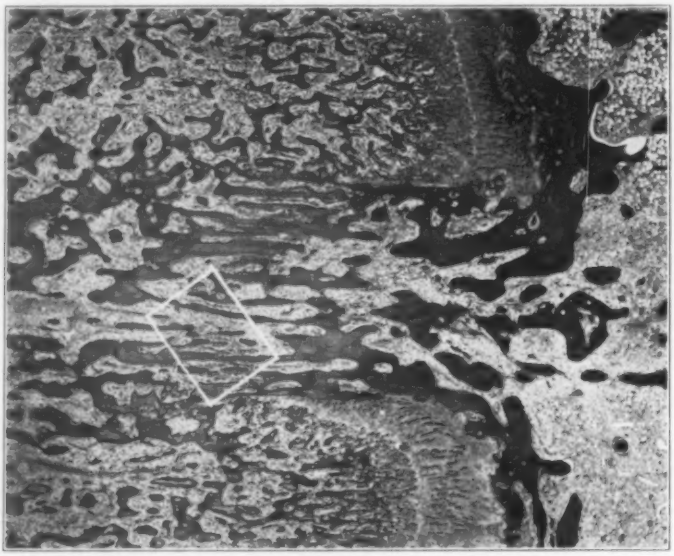

FIG. 11-A

Fibular graft, forty-nine days after implantation. The graft has been replaced almost completely by new bone. The new bone within the drill hole in the plate is certainly as thick as anywhere else, forming a large bridge between the metaphysis and epiphysis. Despite this bridge of bone, growth of the epiphyseal plate is continuing in an essentially normal fashion. Toward the metaphyseal end of the defect (outlined area) the bone trabeculae are thin and oriented in parallel array, much like fish swimming against a current (× 20).

4. Loss of bone continuity: a large gap filled with fibrous tissue, and no suppression of growth of the epiphyseal cartilage (Fig. 3).

In almost all the control femora with drill holes only, there developed some loss of bone continuity in the bone formed in or near the drill hole; little suppression of growth of the epiphyseal cartilage was shown. In all of these specimens there was little evidence of regeneration of the epiphyseal cartilage in the defect or of the formation of a bridge of bone between the metaphysis and epiphysis. In the femora with fibular grafts, the forces of growth and the resistance offered by the bone bridge across the epiphyseal plate seemed more evenly matched because almost half of these tended toward fusion and growth arrest and the remainder showed continued growth. With histological preparations we could evaluate this conflict of forces at all the ages studied.

Markers of silk and carbon were placed one centimeter apart on the fibular

grafts prior to insertion in eight animals ten to twenty-eight days after operation. In six specimens the markers had separated from two to 3.5 millimeters at the time of sacrifice. All of these six femora demonstrated full or partial loss of continuity of the graft. In the other two specimens the markers remained one centimeter apart; both of these femora showed solid fusion across the plate. In general, the gross measurements of the whole femora showed slightly more growth on the control side regardless of whether the graft arrested growth or not. However, at two or three weeks after operation, the difference was usually only one millimeter or less in a femur seven centimeters long.

FIG. 11-B

Higher magnification (× 125) of the area enclosed within the box outlined in Fig. 11-A. At this stage there are bone trabeculae surrounded by osteoblasts, but the intense cellular activity seen in the earlier specimens is not present. Bone formation seems to predominate, with but little evidence of resorption. Despite this, these thin, parallel trabeculae might easily resorb, if stretched. One trabecula (top center) appears to be thinned-out and undergoing resorption. Near the center of another long trabecula (bottom center) an osteoclast seems to be creating a defect at the narrowest part of the trabecula.

MICROSCOPIC EVALUATION

Figures 2 and 3 are examples of solid fusion and complete loss of bone continuity. The other photomicrographs are arranged according to the length of time from operation to sacrifice—from five to forty-nine days. They also show the gradual development of microscopic defects in the bone bridges across the plates. It is the development of these defects that allows growth to continue despite the apparent (grossly) solid bone fusion across the epiphyseal plates.

DISCUSSION

This histological material demonstrates the presence of microscopic losses of continuity in the bone trabeculae traversing drill holes across the growing distal

femoral epiphyseal plates of young rabbits. These losses of continuity could perhaps be called stress fractures, produced by a continuous powerful stretching force. These defects seem to develop gradually by a completely normal physiological process, with cellular absorption and remodeling. As a result, the relatively unstretchable bone is converted at one point to collagenous fibers and connective-tissue cells in various phases of differentiation. This area then elongates in response to the force exerted on it. When this force has been expended by the stretching, we assume that the process reverses itself and the fibers, osteoblasts, and other elements in the area re-form bone. This series of events could possibly occur several times during the period of growth. This breakdown of apparently solid fusions seemed to occur in our fibular grafts twenty days or more after implantation. A majority of the specimens seemed headed for fusion prior to twenty days, whereas after twenty days a large majority showed evidence of loss of continuity.

It is unlikely that the microscopic losses of bone continuity (or stress fractures) demonstrated here would be visible on gross examination or on roentgenograms. This phenomenon can probably be demonstrated only by microscopic study, a fact that would explain some of the confusion and uncertainty found in the literature on the subject.

It has long been thought that stress or fatigue fractures can occur gradually as the result of prolonged stress on a living bone. Perhaps these slides give the first histological evidence of the development of a stress fracture. These fractures occur primarily in the new living bone that replaces the graft and not in the graft itself. In cases in which no graft was used but new bone grew massively across the defect, similar stress fractures of this new bone were seen. These stress fractures appear to be produced by a physiological cellular response and not simply by mechanical rupture of the inorganic framework, such as occurs when a metal bar is broken. In the past such a mechanical rupture has been presumed to occur in most fatigue fractures of human bones.

Since in this study motion has been virtually eliminated as the cause of the fractures or breaks in continuity, it seems probable that the primary cause of the fractures is the tension on the bone produced by the expansion of the epiphyseal plate during growth of the epiphyseal-cartilage cells. This tension has been measured by Strobino and associates as greater than 475 pounds to the square inch in calves; it is not known what the tension is in rabbits, but it assuredly must be great

We do not know the exact mechanism by which these bone defects are formed, that is, what stimulates the osteoclastic activity, what causes the deposition or the dissolving of calcium, or what controls the matrix formation. The technique used in this investigation, however, provides an interesting experimental tool to use in the study of bone tissue under stress. Under these experimental conditions there is pure tension or stretch uncomplicated by many of the factors that interfere with the evaluation of results after grafting procedures in clinical practice. The breakdown, growth, and alignment of bone under such conditions of stress are being studied further.

SUMMARY

Drill holes and fibular grafts were placed across the epiphyseal line of the distal end of the femur in thirty-eight rabbits and drill holes alone were made across the epiphyseal line in thirty-two rabbits. Microscopic sections at various times after surgery showed considerable new bone growing through the defects. When grafts were used they were fused by new bone to the epiphysis and metaph-

ysis. Despite this, in the majority of cases a microscopic defect developed in the bone and epiphyseal growth continued. These bone defects or stress fractures are shown to be accompanied by an intense cellular reaction. The implications of these findings and their relevance to growth after fusion in young human beings are discussed.

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Bone Growth after Spine Fusion

A Clinical Survey

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About 1783, John Hunter placed lead shot two inches apart in the diaphysis of a young pig. When the pig was fully grown and was slaughtered, the shot were still two inches apart. This led him to believe that bones are not elongated by new matter being interposed in the interstices of the old.

For many years the mechanism of longitudinal growth of bone has been a controversial subject in the literature. As eminent an authority as Clopton Havers believed that all parts of the bone tissue of the skeleton took part in longitudinal growth, and that this so-called interstitial growth of bone was the normal form of skeletal growth. Kornew in 1929, Policard in 1930, and Hellstadius in 1947, have all defended some type of interstitial growth of bone.

The majority opinion, especially as roentgenography and microscopy have widened the study of the subject, has been that all growth in length of the diaphyses of long bones takes place at the epiphyseal cartilages, whereas growth of bone in other dimensions occurs through hyaline-cartilage proliferation—as in the epiphyses—or through fibrous-tissue proliferation—as in the periosteum and flat bones. After a careful survey of all arguments for and against this theory, Lacroix concluded that: "The structure of arguments denying the intervention of any interstitial bone growth remains intact".

However, just as the argument seemed settled, new observations arose to confuse the picture. Various investigators reported the apparent lengthening of fused segments after spine fusion in children and in young experimental animals. If this were true in the solidly fused spine, it would mean one of two things: either the fusion area broke down at one time or another or there was interstitial growth of bone in the fusion plate.

On the experimental plane, Bisgard and Musselman performed lateral interbody fusion on the spines of young goats and reported that in every instance fusion by a firm bone bridge resulted. As growth of the spine continued, they noted that this bone bridge became elongated to accommodate itself to the longitudinal growth of the vertebrae. They concluded that under certain circumstances, transplanted bone is capable of intrinsic growth in length.

On the other hand, in the same year, Haas, after performing spine fusions in puppies, noted that just as soon as the fusion was firm in the spinous processes, there was no yielding in the fused spine to the growing forces of the bodies. However, Haas found definite lengthening of the grafted area in almost all of his experiments, but he had no serial roentgenograms, explorations, or other ways to determine when the "fusion was firm". Therefore, the most he could logically infer was that at the time of sacrifice, when the animal was almost fully grown, there was generally solid fusion and that growth had been somewhat suppressed at some time. He noted that when the markers were attached to the graft, there was no separation of the markers. However, this important observation was based on two dogs only.

Odelberg-Johnson [14] performed spine fusions on rabbits, but noted gross pseudarthroses at almost every interspace. He concluded that the growth in length of the spine in these animals took place at these defects. However, these were clinically obvious defects, often with a true joint cavity completely lined with cartilage, and gross motion was often demonstrable in bending roentgenograms. Hence, the findings in these animals are scarcely comparable with those in children with apparently solid spine fusions. In general, therefore, spine-fusion experiments fail to throw much light on the problem because of the difficulty in getting proved solid fusion in young animals.

Clinical reports are as confusing as experimental ones. Hallock, Francis, and Jones, and Risser indicated that growth of the fusion mass does occur. Risser noted that the fusion mass is elongated with growth of the vertebral body and

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1. By our method the centers of the vertebral bodies and mid-points of the pedicles were used as points of reference for all measurements. The two end vertebrae of the fused area were never included in any measurement. Thus, if the fusion extended from the tenth thoracic to the third lumbar vertebra, the distance between the center of the eleventh thoracic vertebra and the center of the second lumbar vertebra would be measured. Similarly, if the fusion extended one segment farther down to the fourth lumbar vertebra, the distance between the center of the body of the eleventh thoracic and that of the third lumbar vertebra would be used as reference points of measurement.

If pseudarthrosis was found between the tenth and eleventh thoracic vertebrae, an alternate measurement would be used. Since the restitution of the diseased area in the upper portion of the body of the first lumbar body is probably equivalent to the encroachment of the inferior surface of the third lumbar vertebra into the adjacent intervertebral disc, the distance between the central point of the body of the first lumbar to the central point of the body of the third lumbar vertebra could be used as an alternate measurement.

Finally, if the fusion extended down one more segment to the fifth lumbar vertebra, measurements could be made in the same fashion from the center of the eleventh or twelfth thoracic or from the center of the first or second lumbar body down to the center of the body of the fourth lumbar vertebra.

Whenever possible, diseased vertebrae were not used as points of reference for measurement.

Measurement of the control area under the conditions described here is illustrated. The points of reference are the centers of the bodies of the seventh and ninth thoracic vertebrae. Note that four vertebral plates are included in the control area being measured.

2. By the method of Hallock, Francis, and Jones, if the fusion area extended from the tenth thoracic to the third lumbar vertebra, the points of reference for the measurement of the length of the whole fusion would be vertebral plate *1* and vertebral plate *10*. Measurements of the normal vertebral bodies included within the fusion would be made from vertebral plate *1* to vertebral plate *4* and from vertebral plate *7* to vertebral plate *10*. Measurements of the normal posterior elements, the pedicles, would be made in a similar fashion between points *A* and *B* and between points *C* and *D* on the pedicles.

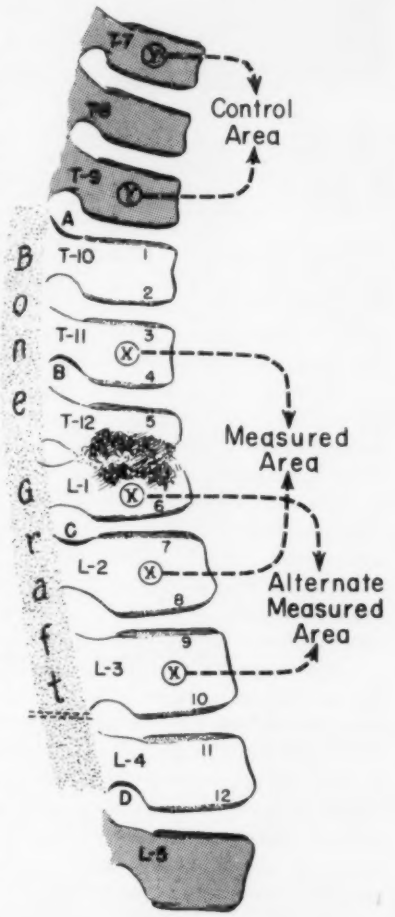

FIG. 1

Two methods of measurement.

FIG. 2-A

FIG. 2-B

Roentgenograms of Case G. C., on whom an Albee fusion was performed, extending from the third to the fifth lumbar vertebra. Pseudarthrosis between the third and fourth lumbar vertebrae appeared six months after operation and was still visible on a roentgenogram made on May 4, 1951, four years after operation (Fig. 2-A). Despite no treatment of any kind, this pseudarthrosis was bridged by bone, May 9, 1952, five years after operation (Fig. 2-B).

concluded that this was possible because of the biological plasticity of bone. Hallock and associates carefully measured the roentgenograms of fifteen Hibbs fusions that were performed in young children who were followed into adult life. These authors found in all patients continued growth of both the anterior and the posterior elements of the fused vertebrae.

Cleveland and associates presented a long-term follow-up study of spine fusions for tuberculosis in children and stated that: "Growth of the fused area was noted, the average growth being one and one-half inches and the least growth being three-quarters of an inch".

Completely opposed to these authors are the findings of Odelberg-Johnson [13], who also made a long-term follow-up of Albee grafts in human beings. He stated categorically that he had not observed increase in length of the whole paraspinal bone bridge in man. Ponseti and Friedman, in discussing changes in the scoliotic spine after fusion, wrote: "The growth of the fused segment, then, was nil or minimal except in the cases in which pseudarthrosis developed".

Another confusing question, raised but not explained in recent literature, is the bending of apparently solidly fused portions of the spine. After spine fusion for scoliosis, von Lackum and Miller observed that bending or giving way of the fusion will occur if the primary curve is overcorrected to the extent that balance of the trunk cannot be restored by straightening of the compensatory curves. Under these circumstances a gradual but sustained loss of correction occurs; occasionally through a localized area of deficient fusion, but often throughout the extent of the immature fusion mass in the absence of any pseudarthrosis, as proved by repeated negative surgical explorations.

Ponseti and Friedman also noted that correction in scoliosis was often lost and that the grafts bent, even when apparently solid. Hallock and Jones, in an earlier paper on tuberculosis of the spine, noted that: "Forward bending with

resultant increasing deformity does not produce a pseudarthrosis in every instance. In many instances it results only in a bending of a somewhat malleable fusion plate. The sites of fusion in thirteen patients were explored for suspected pseudarthrosis, because of increasing deformity, and in each a solid fusion was found".

None of these authors attempted to explain how the bone bends or grows in length, merely stating that bone is "malleable", "plastic", or "ductile". But bone is not malleable, plastic, or ductile; it is rigid, even young bone is rigid. It can alter shape by apposition and absorption; it can grow by cartilaginous or fibrous proliferation; but it cannot grow, stretch, or bend in the mid-portion of a long fusion plate, unless one assumes there is such a thing as interstitial growth, or admits that there is loss of continuity of the bone in certain areas.

(The text begins again on page 1406)

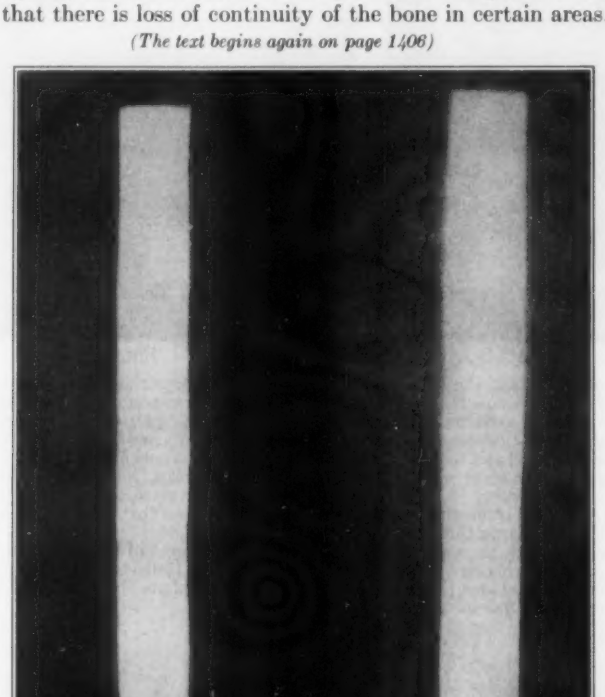

FIG. 3

Roentgenograms of two lead strips illustrating differences in magnification. The distance from the roentgen tube to the cassette was thirty-six inches (79.2 centimeters). Each lead strip was ten centimeters long and one centimeter wide. When this roentgenogram was made the strip on the left (at flat side of ruler) was held 8.5 centimeters above the film (the distance from the film to the spinous processes during a lateral roentgenogram of a six-year-old child, weighing fifty-five pounds) and the strip on the right was held 18.5 centimeters above the film (the distance from the film to the spinous processes during a lateral roentgenogram of an adult of average build weighing 150 pounds). The ruler was placed on the top of the cassette.

On this roentgenogram the strip on the left is 11.1 centimeters in length; the one on the right is 12.8 centimeters in length. Thus, the child's spine is magnified 11 per cent and the adult's spine, 28 per cent. This is a difference in magnification of the two strips amounting to 17 per cent. When the tube-to-target distance was increased to forty inches, as is sometimes the case when making roentgenograms of adult spines, the dimensions of the roentgenogram of the lead strip representing the adult changed very little. It is thus apparent that lateral roentgenograms of an adult's spine are magnified significantly more (17 per cent) in this instance than are those of a child.

FIG. 4-A

FIG. 4-B

Case S. S., tuberculosis of the spine involving the twelfth thoracic and first lumbar vertebrae.

On June 20, 1951, a massive Hibbs fusion, using bank bone, was performed from the ninth thoracic to the third lumbar vertebra. Exploration of the graft with repair of pseudarthrosis between the eleventh and twelfth thoracic vertebrae was carried out on January 9, 1952. On May 21, 1952, the fusion was re-explored and found to be solid throughout its whole extent.

Roentgenograms of the spine were obtained at least once a year, from 1952 to 1958. The best were those made on December 16, 1952, when the patient was five years of age (Fig. 4-A), and on August 9, 1958, when the child was ten and a half (Fig. 4-B). Note that some straightening of the kyphosis had occurred during this interval of five and a half years.

Using these roentgenograms, we determined the increase in height of the fused and the unfused normal vertebrae, employing first our method and then that of Hallock, Francis, and Jones.

By our method the fused area (the six undamaged epiphyseal plates) between the tenth thoracic and second lumbar vertebrae increased as follows:

	12/16/52	7/9/58	Increase (per cent)
Distance between mid-bodies	6.9 cm.	7.5 cm.	9
Distance between mid-pedicles	7.2 cm.	7.9 cm.	10

and the unfused area (six undamaged epiphyseal plates) between the third lumbar and the first sacral vertebrae increased as follows:

	12/16/52	7/9/58	Increase (per cent)
Distance between mid-bodies	6.7 cm.	8.7 cm.	30
Distance between mid-pedicles	6.0 cm.	8.0 cm.	33

By the method of Hallock, Francis, and Jones, the fused healthy vertebrae, measured from the top of the ninth thoracic to the bottom of the eleventh thoracic vertebra and from the top of the second lumbar to the bottom of the third lumbar vertebra, increased as follows:

	12/16/52	7/9/58	Increase (per cent)
T9 to T11	4.7 cm.	5.6 cm.	19
L2 to L3	4.0 cm.	4.9 cm.	22

and the unfused healthy vertebrae, measured from the top of the fourth lumbar vertebra to the bottom of the first sacral vertebra, increased as follows:

L4 to S1	6.0 cm.	8.0 cm.	33 (per cent)

Similarly, by their method, the total fusion area, measured both from the top of the ninth thoracic to the bottom of the third lumbar vertebra and from the superior margin of the pedicle of the ninth thoracic to the inferior margin of the pedicle of the third lumbar vertebra, increased as follows:

	12/16/52	7/9/58	Increase (per cent)
Bodies, T9 to L3	12.0 cm.	13.7 cm.	14
Pedicles, T9 to L3	12.2 cm.	13.5 cm.	11

FIG. 5-A

FIG. 5-B

Case D. R., tuberculosis of the spine involving the fifth and sixth thoracic vertebrae.

On June 16, 1950, a massive Hibbs fusion, reinforced with ribbon grafts from the tibia, was performed from the fourth to the tenth thoracic vertebra.

Roentgenograms at yearly intervals thereafter indicated that the graft was solid throughout its entire extent. The roentgenograms used for measurement were obtained on December 12, 1950, when the patient was eight years old (Fig. 5-A), and on June 2, 1955, when the child was twelve and a half (Fig. 5-B).

Using these roentgenograms, we determined the increase in height of the fused and the unfused normal vertebrae, employing first our method and then that of Hallock, Francis, and Jones.

By our method the fused area (six undamaged epiphyseal plates) between the fifth and ninth thoracic vertebrae increased as follows:

	12/12/50	6/2/55	Increase (per cent)
Distance between mid-bodies	7.3 cm.	8.1 cm.	11
Distance between mid-pedicles	7.5 cm.	8.0 cm.	7

and the unfused area (six normal epiphyseal plates) between the tenth thoracic and the first lumbar vertebrae increased as follows:

	12/12/50	6/2/55	Increase (per cent)
Distance between mid-bodies	7.4 cm.	10.4 cm.	41
Distance between mid-pedicles	7.8 cm.	11.0 cm.	41

By the method of Hallock, Francis, and Jones, the fused healthy vertebrae, measured from the top of the seventh thoracic to the bottom of the tenth thoracic vertebra, increased as follows:

T7 to T10	7.8 cm.	9.4 cm.	21 (per cent)

and the unfused normal vertebrae, measured from the top of the eleventh thoracic to the bottom of the first lumbar vertebra, increased as follows:

T11 to L1	7.2 cm.	10.2 cm.	42 (per cent)

Similarly, by their method, the total fusion area, measured both from the top of the fourth thoracic to the bottom of the tenth thoracic vertebra and from the superior margin of the pedicle of the fourth thoracic to the inferior margin of the pedicle of the tenth thoracic vertebra, increased as follows:

	12/12/50	6/2/55	Increase (per cent)
Bodies, T4 to T10	12.6 cm.	15.1 cm.	20
Pedicles, T4 to T10	12.5 cm.	14.4 cm.	15

FIG. 6-A

FIG. 6-B

Case B. A. S., tuberculosis of the spine involving the third and fourth lumbar vertebrae.

On February 17, 1950, a spine fusion from the first lumbar to the fifth lumbar vertebra was attempted, but pseudarthroses developed between the third and fourth and between the fourth and fifth lumbar vertebrae. On February 9, 1951, these pseudarthroses were repaired successfully. However, a pseudarthrosis that had been overlooked in several roentgenograms was subsequently seen between the first and second lumbar vertebrae.

Yearly roentgenograms thereafter revealed that the graft was solid from the second lumbar to the second sacral vertebra. The roentgenograms used for measurement by both our method and that of Hallock, Francis, and Jones were obtained on June 15, 1951, when the child was seven years old (Fig. 6-A), and on April 11, 1958, when the child was fourteen (Fig. 6-B).

By our method the fused area (four undamaged epiphyseal plates) between the fourth lumbar and first sacral vertebrae increased as follows:

	6/15/51	4/11/58	Increase (per cent)
Distance between mid-bodies	5.7 cm.	6.2 cm.	9
Distance between mid-pedicles	4.9 cm.	5.3 cm.	8

and the unfused area (four normal epiphyseal plates) between the eleventh thoracic and the first lumbar vertebrae increased as follows:

	6/15/51	4/11/58	Increase (per cent)
Distance between mid-bodies	5.2 cm.	7.3 cm.	40
Distance between mid-pedicles	5.5 cm.	7.3 cm.	33

By the method of Hallock, Francis, and Jones, the fused healthy vertebrae, measured from the top of the second lumbar to the bottom of the third lumbar vertebra, increased as follows:

L2 to L3	4.8 cm.	6.2 cm.	29 (per cent)

and the unfused normal vertebrae, measured from the top of the twelfth thoracic to the bottom of the first lumbar vertebra, increased as follows:

T12 to L1	4.8 cm.	6.8 cm.	42 (per cent)

Similarly, by their method, the total fusion area, measured both from the top of the second lumbar to the bottom of the first sacral vertebra and from the superior margin of the pedicle of the second lumbar to the inferior margin of the pedicle of the first sacral vertebra, increased as follows:

	6/15/51	4/11/58	Increase (per cent)
Bodies, L2 to S1	12.5 cm.	15.0 cm.	20
Pedicles, L2 to S1	11.5 cm.	13.5 cm.	17

FIG. 7-A

FIG. 7-B

Case M.L., tuberculosis of the spine involving the third, fourth, and fifth lumbar vertebrae.

An Albee fusion from the first lumbar to the first sacral vertebra was attempted on August 14, 1942, when the patient was four years old. Pseudarthroses developed between the second and third and between the third and fourth lumbar vertebrae. These pseudarthroses were repaired on January 26, 1943.

Yearly roentgenograms thereafter revealed the fusion to be solid from the first lumbar to the second sacral vertebra. The roentgenograms used for measurement by both our method and that of Hallock, Francis, and Jones were obtained on July 1, 1943, when the patient was five years old (Fig. 7-A), and on August 25, 1949, when the child was eleven (Fig. 7-B).

By our method, the fused area (four undamaged epiphyseal plates) between the second lumbar and first sacral vertebrae increased as follows:

	7/1/43	8/25/49	Increase (per cent)
Distance between mid-bodies	6.2 cm.	6.8 cm.	10
Distance between mid-pedicles	6.3 cm.	6.9 cm.	10

and the unfused area (four normal epiphyseal plates) between the tenth and twelfth thoracic vertebrae increased as follows:

	7/1/43	8/25/49	Increase (per cent)
Distance between mid-bodies	4.2 cm.	5.9 cm.	40
Distance between mid-pedicles	4.3 cm.	5.8 cm.	35

By the method of Hallock, Francis, and Jones the fused healthy vertebrae, measured from the top of the first lumbar to the bottom of the second lumbar vertebra, increased as follows:

L1 to L2	4.5 cm.	6.0 cm.	33 (per cent)

and the unfused normal vertebrae, measured from the top of the tenth to the bottom of the twelfth thoracic vertebra, increased as follows:

T10 to T12	6.0 cm.	8.5 cm.	42 (per cent)

Similarly, by their method, the total fusion area, measured both from the top of the first lumbar to the bottom of the first sacral vertebra and from the superior margin of the pedicle of the first lumbar to the inferior margin of the pedicle of the first sacral vertebra, increased as follows:

	7/1/43	8/25/49	Increase (per cent)
Bodies, L1 to S1	10.5 cm.	12.3 cm.	17
Pedicles, L1 to S1	10.0 cm.	11.5 cm.	15

FIG. 8-A

FIG. 8-B

Case G.N., tuberculosis of the spine involving the sixth, seventh, and eighth thoracic vertebrae. A two-stage (the first on December 8, 1950, and the second on January 26, 1951) Hibbs fusion from the third thoracic to the twelfth thoracic vertebra was attempted, using bank bone. Fusion was solid from the third to the eleventh thoracic vertebra, as evidenced by a roentgenogram made in April 1951, but there was pseudarthrosis between the eleventh and twelfth thoracic vertebrae.

Roentgenograms were obtained frequently during the following two years. However, there was then a two-year lapse; the next roentgenogram was not made until 1955. At this time there was evidence that the pseudarthrosis had healed spontaneously, without treatment. Measurements by both our method and that of Hallock, Francis, and Jones were made on the basis of the initially solid fusion extending from the third to the eleventh thoracic vertebra. The roentgenograms used were obtained on August 6, 1951, when the patient was five years old (Fig. 8-A), and on June 7, 1955, when the child was nine (Fig. 8-B). In the interval between these roentgenograms the kyphosis had diminished slightly.

By our method, the fused area (eight undamaged epiphyseal plates) between the fourth and tenth thoracic vertebrae had increased as follows:

	8/6/51	6/7/55	Increase (per cent)
Distance between mid-bodies	8.5 cm.	8.7 cm.	2
Distance between mid-pedicles	10.0 cm.	10.2 cm.	2

and the unfused area (eight normal epiphyseal plates) between the first and fifth lumbar vertebrae had increased as follows:

	8/6/51	6/7/55	Increase (per cent)
Distance between mid-bodies	10.5 cm.	12.2 cm.	16
Distance between mid-pedicles	9.9 cm.	11.0 cm.	11

By the method of Hallock, Francis, and Jones, the fused healthy vertebrae, measured from the top of the ninth thoracic to the bottom of the eleventh thoracic vertebra and from the top of third thoracic to the bottom of the fifth thoracic vertebra, increased as follows:

	8/6/51	6/7/55	Increase (per cent)
T9 to T11	5.3 cm.	6.0 cm.	13
T3 to T5	4.7 cm.	5.2 cm.	11

and the unfused normal vertebrae, measured from the top of the first lumbar to the bottom of the third lumbar vertebra, increased as follows:

L1 to L3	7.1 cm.	8.1 cm.	14 (per cent)

Similarly, by their method, the total fusion area, measured both from the top of the third thoracic to the bottom of the eleventh thoracic vertebra and from the superior margin of the pedicle of the third thoracic to the inferior margin of the pedicle of the eleventh thoracic vertebra, increased as follows:

	8/6/51	6/7/55	Increase (per cent)
Bodies, T3 to T11	12.0 cm.	12.9 cm.	7
Pedicles, T3 to T11	13.1 cm.	14.0 cm.	7

FIG. 9-A

FIG. 9-B

Case W. R., tuberculosis of the spine involving the fourth and fifth lumbar vertebrae.

On February 4, 1949, a Hibbs fusion, reinforced with tibial bone, was performed from the first lumbar to the second sacral vertebra. Fusion became solid from the second lumbar to the second sacral vertebra, with pseudarthrosis between the first and second lumbar vertebrae.

Thereafter, roentgenograms were obtained at least once yearly until 1955. The roentgenograms used for measurement by both our method and that of Hallock, Francis, and Jones were obtained on June 7, 1949, when the patient was seven years old (Fig. 9-A), and on June 6, 1955, when the child was thirteen (Fig. 9-B).

By our method, the fused area (four undamaged epiphyseal plates) between the third lumbar and first sacral vertebrae increased as follows:

	6/7/49	6/6/55	Increase (per cent)
Distance between mid-bodies	6.7 cm.	7.1 cm.	6
Distance between mid-pedicles	6.2 cm.	6.4 cm.	3

and the unfused area (four normal epiphyseal plates) between the tenth and twelfth thoracic vertebrae increased as follows:

	6/7/49	6/6/55	Increase (per cent)
Distance between mid-bodies	5.1 cm.	6.5 cm.	25
Distance between mid-pedicles	5.3 cm.	6.8 cm.	28

By the method of Hallock, Francis, and Jones, the fused healthy vertebrae, measured from the top of the second lumbar to the bottom of the third lumbar vertebra, increased as follows:

	6/7/49	6/6/55	Increase (per cent)
L2 to L3	5.5 cm.	6.7 cm.	22

and the unfused normal vertebrae, measured from the top of the tenth thoracic to the bottom of the twelfth thoracic vertebra, increased as follows:

T10 to T12	7.0 cm.	9.1 cm.	30 (per cent)

Similarly, by their method, the total fusion area, measured both from the top of the second lumbar to the bottom of the first sacral vertebra and from the superior margin of the pedicle of the second lumbar to the inferior margin of the pedicle of the first sacral vertebra, increased as follows:

	6/7/49	6/6/55	Increase (per cent)
Bodies, L2 to S1	11.7 cm.	12.8 cm.	9
Pedicles, L2 to S1	10.3 cm.	11.0 cm.	7

Epiphyseal growth will be suppressed in an area of increased pressure and more active in an area of decreased pressure—according to the Hueter-Volkmann law—thus allowing bending through growth. But the shaft of a long bone or a long fusion plate responds to Wolff's law, which states that increased pressure causes increased bone formation and consequent greater strength of the bone in the area of stress, with the result that bending does not occur. There is, however, another law. Weinmann and Sicher stated that: "Increase of pressure or tension beyond the limits of tolerance leads to destruction of bone by resorption". This obviously occurs with pressure caused by certain tumors and aneurysms. Could not this also be true in spine fusions, in which excessive tension, due to growth, or excessive pressure, due to overcorrection of scoliosis, may produce resorption of bone sufficient to cause an actual loss of continuity of the graft in one area? Th s loss of continuity, pseudarthrosis, or stress fracture—call it what you will —may then allow stretching or bending of the graft.

CLINICAL INVESTIGATION

This study was initiated because of the clinical problem of young children requiring long spine fusions for such diverse reasons as idiopathic scoliosis, tuberculosis, poliomyelitis, and congenital anomalies. It is certainly important, before subjecting a small child to such an operation, to know the long-term, as well as the short-term effects of the procedure. An accurate knowledge of bone growth after fusion is essential to evaluate whether, in a given situation, the desirable effects of the operation will outweigh any possible long-term undesirable effects. Such knowledge is also vital in determining the extent and, perhaps, the type of fusion.

Although the recent majority opinion, as expressed in the literature, seems to be that growth still persists after fusion, at a slower but a substantial rate, it seemed to us that this concept is contrary to the basic laws of bone physiology. Therefore, it was decided to evaluate the clinical material at the Children's Hospital and the Kernan Hospital for Crippled Children, both in Baltimore, to see what happened in actual situations. A wealth of clinical material was available and there were many long-term follow-ups.

Attempts to determine the growth of the fusion mass after spine fusion for scoliosis were unrewarding. It is almost impossible to judge by roentgenogram the solidity of a fusion in a thoracic spine with scoliosis. Furthermore, if the curvature and rotation are significant, accurate measurement of the length of the fusion area is extremely difficult.

Hibbs and Albee fusions for tuberculosis of the spine in children under ten years of age were next studied. Roentgenograms of the spines of 400 children with tuberculosis of the spine were available in the two hospitals, yet it was amazing how few could be properly evaluated with respect to bone growth after fusion. In many cases, it was extremely difficult to judge the solidity of the fusion, especially the Hibbs fusion in the thoracic region. In spines in which the fusion plate could be clearly seen, the incidence of pseudarthrosis in one or more areas was almost 75 per cent. Often, these pseudarthroses were near the ends of the fusion and they often healed spontaneously, as shown by later roentgenograms. These defects did not seem to interfere greatly with the healing of the tuberculosis, but they did interfere with growth evaluation. Many cases had incomplete follow-up roentgenograms and records. Finally, the long-term follow-up roentgenograms—twenty years or more after fusion—seemed so grossly magnified and of such different technique that it was extremely difficult to make accurate comparisons.

With these problems in mind, it was decided to review the paper by Hallock, Francis, and Jones more closely, since this is the only one that gives in detail the criteria and techniques used for growth measurements after spine fusion.

Their patients were all treated by Hibbs fusion; no mention is made of additional bone grafts being taken from other areas. Seven fusions were in the thoracic area, and eight were in thoracolumbar or lumbar areas. Kyphosis was moderate to moderately severe in thirteen of fifteen patients, thus making accurate measurement difficult. They stated that fusion was successful in all patients but they did not mention what roentgenographic criteria were used to determine this, or if the presence of fusion was verified by surgical exploration. Measurements were made from "early postoperative" roentgenograms, but the exact time after operation was not stated.

Their measurements were of the normal vertebrae included within the extent of the fusion and of the whole fusion area. The normal vertebrae, usually two above or below the diseased area, were measured from the top of the top vertebral body to the bottom of the bottom vertebral body (as from *1* to *4* in Fig. 1). Thus, the end one of the four epiphyseal plates included in the region measured (Fig. 1, *1*) is completely unaffected by the fusion, and hence at least 25 per cent of the normal growth of the segments being measured could be expected to occur. At the other end of the measured segments (Fig. 1, *4*), the disc space in the roentgenograms of older patients is markedly narrowed, as noted by Hallock, Francis, and Jones, and this narrowing probably occurs because of bone replacement of the disc material. At any rate, the disc space is largely replaced by the adjacent vertebral bodies and any measurement using the vertebral end-plate in this way would show growth of the individual vertebral body, but this growth may be at the expense of the disc and does not indicate growth of the fused segment as a whole.

Measurement of the whole fused area (Fig. 1, from *1* to *10* or from *1* to *12*, depending on the length of fusion) will include two growth plates (two out of ten or twelve) that are not restricted by the fusion. Hence, 15 to 20 per cent of the normal growth of these six or seven fused vertebrae will take place anyway, not in the least influenced by the fusion. Measurement of the distance between the pedicles is subject to the same consideration, although to a lesser degree. The uppermost or lowermost pedicles are free to grow in width, especially if the adjacent vertebral body grows and pulls the periosteum of the pedicles with it. In a fused area the bone of the pedicle may expand somewhat at the expense of adjacent ligaments.

The presence of pseudarthrosis was never mentioned as a possibility by Hallock, Francis, and Jones. In our series, numerous defects in the fusion plates were found on close examination. Sometimes, these were not visible in one roentgenogram, especially the one made early after operation, but were found on subsequent roentgenograms. In other instances they were present in the early roentgenograms, but subsequently healed spontaneously (Fig. 2). A study of numerous roentgenograms of the same fusion over a long period of time is necessary to rule out adequately either the presence or the absence of defects in the fusion.

Finally, the ages of the children in the early postoperative roentgenograms are not given by Hallock, Francis, and Jones. Presumably, these children were from four to six years old and probably weighed about fifty pounds. The average follow-up was twenty-one years; hence, the follow-up roentgenograms used for comparison were of adults who presumably weighed about 150 pounds. The magnification factor to be expected from these, if the tube-to-target distances were the same (thirty-six inches), would be 17 per cent (Fig. 3).

To illustrate, let us suppose that a fused area during growth increases from 2.8 to 3.4 centimeters and an unfused area from 2.4 to 4.1 centimeters, as shown by roentgenograms made first when the patient was a small child and later when he was an adult. By using the correction factors shown in Figure 3, the childhood measurements are enlarged 11 per cent. When corrected, these will be changed from 2.8 to 2.52 centimeters and from 2.4 to 2.16 centimeters. The adult measurements are enlarged 28 per cent and hence, when corrected, will be changed from 3.4 to 2.65 centimeters and from 4.1 to 3.2 centimeters. Thus, after correction for magnification, we find that the fused area has grown from 2.52 to 2.65 centimeters, or 4 per cent growth, and the unfused area from 2.16 to 3.2 centimeters, or 45 per cent growth. Now, 0.13 centimeter, or 4 per cent growth, is statistically insignificant and well within the limits of error in measurement, so for practical purposes no significant growth has taken place in the fused area, and about 45 per cent growth has taken place in the unfused area.

Using these same figures Hallock, Francis, and Jones estimated that the fusion area grew 120 per cent, a change of 20 per cent, and that the normal area grew 170 per cent, a change of 70 per cent. Using these values, these authors calculated the growth by subtracting one value from the other. The result was 50 per cent less growth in the fusion area; whereas, in point of fact, there is no real evidence that the fusion area grew at all. The only evidence provided by these figures is that the unfused portion grew somewhat in comparison with the fused area.

METHOD

In our series an attempt was made to avoid the variable factors just mentioned by setting up a rigid set of criteria. First to be included was that a solid fusion plate had to be clearly visible on the roentgenograms. This ruled out most Hibbs fusions, unless extensive new bone had been added. Albee operations in the lumbar and thoracolumbar area were the most easily evaluated. All cases with pseudarthroses in the measured area were discarded, and no cases were considered unless the fusion plate was clearly visible throughout the measured area.

The second criterion was that the first measurement should not be made until at least four months after surgery. A graft takes several months to become solidly incorporated; growth may occur during this interval. An Albee graft, in particular, may look solid shortly after surgery but may break down later. A spine measured shortly after surgery and again many years later may look solid on both sets of roentgenograms and yet have had numerous pseudarthroses in the interval.

The third criterion was that all patients should have had numerous roentgenograms, preferably once a year, during the period of measurement to be sure that the fusion had remained solid the entire time. This requirement was instituted because a number of cases were found in which pseudarthroses had developed and then had healed spontaneously and also because some roentgenograms for technical reasons showed no defects, whereas other roentgenograms made before and after these had demonstrated defects.

The fourth criterion was that the fusion area to be measured should include at least four undamaged vertebral epiphyseal plates, all well within the limits of the fusion mass, and that the length of this fused area be compared with that of an unfused portion of the spine containing an equal number of growth plates. The fused area being measured should not contain an end vertebra since such a measurement would include the growth of an end-plate unrestrained by the fusion. Furthermore, no measurements should be made from the end-plates since meas-

urements from these points of reference might include the unrestrained growth of the top and bottom vertebral plates which takes place at the expense of the adjacent intervertebral discs. Instead, measurements should be made from the mid-points of the top and bottom vertebral bodies since these points would be expected to remain relatively constant with bone replacement of the intervertebral discs above and below these central points being about equal (Fig. 1). These measurements sometimes spanned the disease area, but the disease was quiescent in all cases and restitution of the involved area, rather than further destruction, was the rule.

Measurements of the posterior elements were made from the mid-points of the pedicles, as visualized in the lateral roentgenogram rather than from the upper and lower borders of the pedicles. In the absence of markers other posterior measurements were grossly inaccurate, and even these measurements between the pedicles were not so accurate as those between the centers of the vertebral bodies.

The fifth criterion was that the follow-up period should be long enough to show significant growth in the unfused areas, but not too long, because the physique of the child may change radically and cause gross differences in the magnification of the roentgenograms. Only roentgenograms made in the modern era (since 1940) were used to make sure that the roentgenographic techniques were fairly well standardized. Spines with excessive kyphoses or scolioses were avoided, since these deformities make measurement difficult; only one of the cases finally accepted for study had more than a mild kyphosis. A range of follow-up of from four to eight years was approved since, with yearly roentgenograms made during this interval, it was possible to follow the gradual growth of the spine and thus avoid undue distortion. Undoubtedly, some degree of magnification occurred and the roentgenographic techniques and positioning of the patient were not completely standardized, but the roentgenograms were as nearly comparable as could be obtained without some constant factor, such as a one-centimeter metal marker embedded in the graft. It is hoped that future series will be conducted with such a marker system to avoid the variable factors that render interpretation so difficult at present.

These criteria were so exacting that it was necessary to discard, for one reason or another, the great majority of cases reviewed. It was possible finally to collect only six cases from the two hospitals which fulfilled all the criteria mentioned. These cases were measured by our own system and also by that of Hallock and associates. The results of these measurements are recorded in the legends for the reproduction of the roentgenograms used in this study. The photographs of the roentgenograms were made at the same distance and magnification for each pair so that measurements of these photographs should give the same percentage differences as the original roentgenograms.

RESULTS

The average percentages of growth of the fused and unfused vertebrae in the six cases studied were determined by two methods.

1. Our criteria: mid-body measurements of the fusion area, 8 per cent; mid-body measurements of the normal area, 32 per cent; mid-pedicle measurements of the fusion area, 7 per cent; and mid-pedicle measurements of the normal area, 30 per cent.

2. Hallock, Francis, and Jones' criteria: normal fused vertebrae, measured from the top of the top vertebra to the bottom of the bottom vertebra, 21 per cent; normal unfused vertebrae, measured from the top of the top vertebra to the bottom of the bottom vertebra, 34 per cent; total fusion area, measured from

the top of the top vertebra to the bottom of the bottom vertebra, 14 per cent; and total fusion area measured from the superior margin of the pedicle of the top vertebra to the inferior margin of the pedicle of the bottom vertebra, 12 per cent.

It is apparent that even without any correction for magnification or distortion, the growth, as measured by our criteria, was only 25 per cent as much by the mid-body measurements and 23 per cent as much by the mid-pedicle measurements in the fused areas as in the unfused areas. With the criteria of Hallock and associates used for the measurement of these same spines, the normal vertebrae included in the fusion grew 62 per cent as much as the normal vertebrae outside the fusions. The whole fusion area from top to bottom grew 41 per cent as much anteriorly in the vertebral bodies and 35 per cent as much in the region of the pedicles as did the adjacent unfused areas.

Hallock, Francis, and Jones did not specifically state that the rate of growth in the fused area was a certain percentage of the normal rate. However, their statement that the vertebral bodies of the entire fusion area grew 37 per cent less than the normal vertebral bodies seems to imply that the vertebral bodies within the fused area grew 63 per cent as much as the normal vertebral bodies. Similarly, if the normal vertebral bodies within the fusion area grow 23 per cent less than normal vertebral bodies, the inference is that their growth was 77 per cent of the normal. We believe that in future studies of vertebral growth every effort should be made to reduce distortion and magnification to a minimum and then make a direct comparison, admitting that a certain degree of inaccuracy still persists.

The cases in this series are not directly comparable with those in the series studied by Hallock and associates in that the follow-up period in our series is shorter (for reasons mentioned previously). However, the percentage of growth should be relatively comparable, whether the follow-up is five or fifteen years. When Hallock, Francis, and Jones' technique for the measurement of vertebral growth was used in this series, the amount of growth of the vertebral bodies in the whole fusion area was 41 per cent of normal. In the series reported by Hallock and associates, the amount of growth in the fused area of their patients was, by inference, 63 per cent of normal. Similarly, the amount of growth of the normal vertebral bodies included in the fusion area was 62 per cent of normal in our series and 77 per cent of normal, by inference, in the series studied by Hallock, Francis, and Jones.

Although these results are not too far apart, the rate of growth in our series was definitely smaller than that in the other series. We believe that the explanation for this difference is that intercurrent pseudarthroses have been carefully excluded in our cases. Among the fifteen cases studied by Hallock and associates, the amount of growth of the fused normal vertebrae was 100 per cent of normal in two, less than 50 per cent in three, and between 50 and 100 per cent in the remaining cases. This rather wide variation in the amount of growth in one series suggests some difference in the restraining force exerted by the fusion plate—a difference that could well be explained by unrecognized transitory pseudarthroses.

SUMMARY AND CONCLUSIONS

It was concluded from these cases that when a spine fusion is unquestionably solid and fairly massive, there is little increase in length of the fused area. The small increase that most of the cases we studied showed could be accounted for by magnification and other technical factors, but it is impossible to rule out a small amount of growth. The slight decrease in the kyphos in two of our cases suggests some bending of the fusion mass. No definite pseudarthroses could be

seen on the roentgenograms but the presence of one or more pseudarthroses could not be ruled out. Microscopic and transient pseudarthroses are considered by us to be the most likely mechanism by which any real increase in length or any true change in angulation occurs. In our experimental studies [9], microscopic losses of bone continuity in transepiphyseal bone grafts in the distal femora of young rabbits were demonstrated. These defects were of such a nature that they could not be demonstrated by standard clinical or roentgenographic methods.

In our opinion, the end-result study of Hallock and his associates is valuable in that it indicates, from the practical, clinical point of view, what will happen to the average patient after spine fusion in early childhood. However, their data and the observations of the other authors previously mentioned convey an inaccurate and perhaps unintentional impression that considerable growth occurs in a solidly fused spine segment. It would be unfortunate, we believe, to allow this impression to persist since surgeons not familiar with all that is known about growth of the spine after spine fusion might be falsely encouraged on the basis of published data to perform longer and more massive spine fusions in young children. Our study suggests that a long, massive, and completely solid fusion in early childhood will impair spine growth to a significant degree.

We believe that growth of a fused segment of the spine can occur only at the ends of the segment or at the site of gross or microscopic defects in the fusion plate. Pseudarthroses in spine fusions in children are much more frequent, in our opinion, than is generally suspected because of the tension forces exerted by the growing epiphyseal cartilages, as well as the usual stresses caused by motion. These pseudarthroses or stress fractures may be microscopic or grossly visible; they may occur spontaneously at any time and heal spontaneously. The more massive the fusion plate, the less chance there will be that it will break down under the stress of growth and motion. Finally, we believe that the laws that govern bone growth in general apply to the bone of spine fusions. There is in our opinion no such thing as interstitial growth of bone.

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The Problem of the Primary Curve*

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Scoliosis has always been one of the more difficult problems in orthopaedics. It probably was in André's mind when he designed the symbol of orthopaedics. The word *scoliosis* derived from the Greek *skolios*, meaning crooked, does not really indicate the problem.

Albert Ferguson's concept of a primary curve was, in my opinion, the first great step in the solution of the problem of scoliosis. He was the first to teach that a curve or curves may be the result of a deforming factor or factors, whereas another curve or curves in the same patient are a physiological adjustment to maintain balance and bring, if possible, the head erect over the pelvis or bring the eyes horizontal. He defined a primary curve as one produced by a deforming factor or force. He believed that a secondary curve was the result of the action of the law of balance and that muscles act involuntarily to bring the head erect over the center of the pelvis with the body in balance, in the easiest possible way and to the extent that the muscles are able.

Ferguson shows us that scoliosis is not just a crooked spine but a deformity of certain vertebral segments, resulting in a curve or curves which can be localized, measured, and treated. Without Ferguson's concept of a primary curve the excellent results that are now obtained by spine arthrodesis would not be possible.

Unfortunately, Ferguson's concept of a primary curve has not been understood by many; the term has been misused and a new problem has arisen. We still hear the question, "Which is the primary curve?"

For a while there was talk of "the primary curve". Then we heard talk of "the double primary curve". This should have made it clear that there may be a number of primary curves resulting from a deforming factor or factors. It should have been clear that, with only twenty-four movable vertebral segments, when there are quadruple or quintuple primary curves there will be few movable vertebral segments available to form a secondary curve.

Apparently, many orthopaedists who talk about scoliosis have never really understood Ferguson's concept of a primary curve. Only recently, the director of a large scoliosis clinic told me that the primary curve came first and the other curves came as a result of it and were, therefore, secondary. Evidently, to him and many others there is a primary curve which comes first and, as a result of this, other opposite secondary curves develop for compensation. Although this concept is tenable, it ignores the possibility and probability that there may be some process or force resulting in a number of primary curves of different degree.

I am sure that for Ferguson, Risser, and those who understand Ferguson's concept of a primary curve and a secondary curve, the primary curve is no problem. However, so many orthopaedists express doubt or concern about the primary curve that it seems desirable to explain why it should be no problem.

THE NORMAL SPINE

The normal spine and its normal curves suggest terms and definitions for use in the study of abnormal spinal curves. In the fetus there is usually an anterior (flexion) angulation of most, or all, of the vertebral segments. In the postnatal

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period, opposite or posterior (extension) angulation must occur for the individual to assume the erect posture with the head level and centered over the sacrum. Since there is no mobility in the sacrum and limited mobility (especially posterior angulation) in the thoracic vertebrae, this necessary posterior vertebral angulation naturally occurs in the normally more flexible lumbar and cervical regions where mobility (and especially posterior angulation) is greatest. This normal posterior angulation of vertebral segments in the lumbar and cervical areas thus produces curves we call lordosis; these compensate for the normal vertebral angulations that produce the anterior curves or kyphosis in the sacral and thoracic regions.

If, in the erect position, we did not have these normal compensatory opposite posterior angulations or curves, the head would be angulated anteriorly, as occurs in certain patients with Marie-Strümpell arthritis who have a fixed total anterior angulation and no mobility in the lumbar and cervical areas for opposite (posterior) compensatory angulation.

Thus, in the normal erect human spine in the anteroposterior plane (or lateral view) we have four normal spinal curves—two concave anteriorly (sacral and thoracic) and two concave posteriorly (lumbar and cervical)—instead of the single anterior concave curve or the prenatal type. These four normal curves in the anteroposterior plane of the erect human spine vary a great deal in structure and function. The sacral segments, angulated anteriorly, are unsegmented and because of their structure do not have the function of mobility, such as angulation and rotation. We might, then, call the normal curve of the sacrum a *structural curve* since it is a curve caused by the structure of the segments in that area; one might consider this a primary curve. The vertebrae in the lumbar and cervical areas are, in the erect spine, angulated posteriorly, forming spinal curves concave posteriorly and, since mobility such as angulation and rotation is a normal function of these portions of the spine, these normal, flexible, correctable spinal curves may be called *functional curves;* their characteristics fit Ferguson's definition of secondary curves.

The structure of the thoracic vertebrae allows a certain amount of functional mobility but does not usually permit the normal thoracic curve to straighten completely. This normal thoracic curve has some structural and some functional elements and is an example of a combined structural-functional curve.

Thus in the anteroposterior plane (lateral view) of the erect human spine we have examples of three types of curves: (1) structural, (2) functional, and (3) combined structural-functional.

Although in the normal erect spine there are these curves in the anteroposterior plane, there are no lateral curves. However, the normal function of the lumbar, thoracic, and cervical vertebrae permits lateral bend or angulation and some rotation in these areas. These lateral curves, which accompany side bending and result from the normal lateral angulation of vertebral segments, always straighten when the person is erect. Since they result from normal vertebral function, they may be called functional lateral curves. The amount of lateral bend or angulation varies considerably in these three areas (cervical, thoracic, and lumbar), there being much more normal lateral bend or angulation possible in the lumbar and cervical areas than in the thoracic area. Also, the relatively slight lateral bend or intervertebral angulation that is possible in the thoracic area occurs mainly between the lower six thoracic segments with very little between the upper six.

Whenever a person bends to one side (or either direction in any plane) a number of vertebrae angulate in relationship to each other and, being multiple

adjacent segments angulated in the same direction, they geometrically form a curve. In this functional type of curve all of the segments in the curve are angulated to the side of the concavity of the resultant curve and the position of each segment is displaced from the central spinal axis to the side of the concavity, the top segment of the curve being the most displaced.

Thus a normal, functional, lateral, spinal curve has certain definite characteristics apparent on a single erect anteroposterior roentgenogram.

THE ABNORMAL SPINE

When we consider the erect spine with multiple lateral curves (the usual situation) we realize that, just as in the anteroposterior plane of the normal spine, these curves may be structural, functional, or combined structural-functional. We know there should be no lateral curves in the normal erect spine so at least one of the curves must be a structural curve (due to some structural change or abnormality in bone, nerve, muscle, or other tissues). The problem then is: "What are the relative structural and functional elements in the curves and which are the major structural curves?"

From the study of the normal erect spine, we know the main characteristics of a normal functional lateral curve and how such a curve varies in different parts of the spine. The significant signs of structural changes in a curve may be found by the study of the mechanics of any erect segmented column.

In any segmented column, deformities may be caused by abnormal angulation, rotation, or position of one or more segments, or by any combination of these. However, angulation causes the greatest displacement of the capital from its normal level position on a central axis over the base. Greater displacement occurs if multiple adjacent segments are angulated in the same direction in the same plane so that geometrically they form a curve.

In the spinal column any curve is the geometric result of the total angulation (segmental or intersegmental) of a number of adjacent vertebral segments in one direction in one plane. The greater the total angulation in one direction, the greater the displacement of the head in that direction away from its normal position, level and centered over the sacrum.

From the mechanics of any segmented column, including the spinal column, we see then that any abnormal angulation, rotation, or position of vertebral segments is a sign of a structural change.

When we study the anteroposterior roentgenogram of an erect spine with lateral curves there is usually some lateral wedging in the segments of one or omre of these curves. This wedging or segmental angulation is not found in the normal vertebrae and is therefore another definite sign of structural change in the curve.

Thus any abnormal wedging, angulation, rotation, or position of vertebrae in a lateral spinal curve is a sign of structural change. The relative amount of these changes may be measured and expressed as mild (+), moderate (2+) or severe (3+). Any significant amount of one or more of these signs gives us an estimate of the structural changes in a given curve.

ROENTGENOGRAPHIC CHARACTERISTICS

The anteroposterior roentgenogram of the normal erect spine, the normal spine with a functional lateral curve, and a spine with a structural curve shows certain vertebral characteristics.

On the anteroposterior roentgenogram, the vertebrae in the normal erect spine show no wedging, angulation, or rotation, and the position of each vertebra is on a central axis.

When the normal spine is bent laterally there is no vertebral wedging but

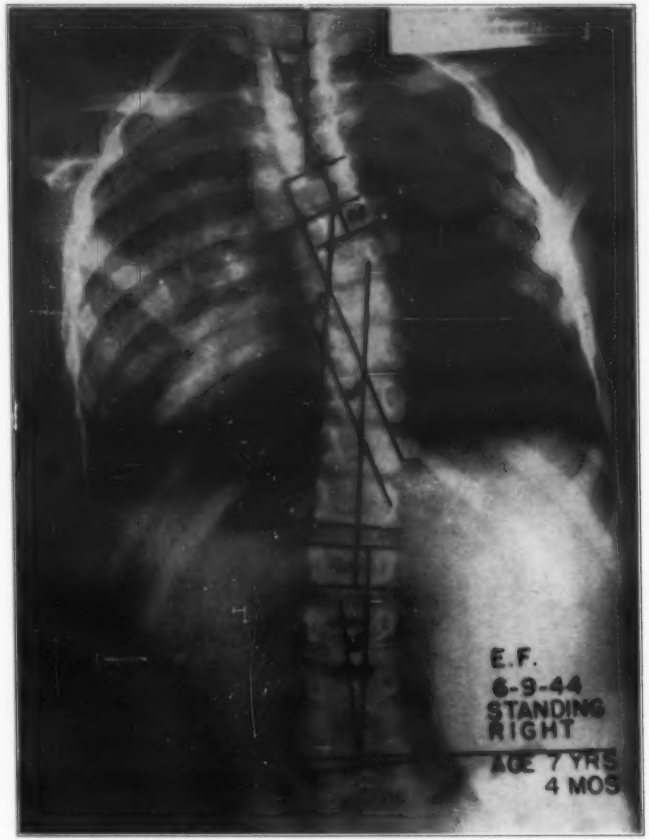

FIG. 1-A

FIG. 1-B

Fig. 1-A: E. F. Three mild curves. There are signs of structural changes in the upper two curves (abnormal wedging, rotation, and position of the vertebrae).
Fig. 1-B: After two years of preventive treatment (cast, brace, and exercises). The upper two curves are structural (abnormal wedging, angulation, rotation, and position of vertebrae). The lower curve shows no significant structural signs.

FIG. 1-C

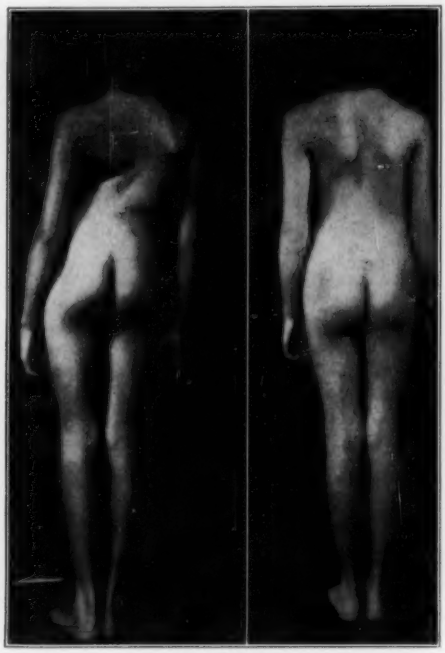

FIG. 1-D

Fig. 1-C: One year after arthrodesis. No increase in lower functional curve.
Fig. 1-D: Before and after arthrodesis.

there is angulation of a number of adjacent vertebral segments forming a smooth curve which is more in the lumbar than in the thoracic area. Associated with this angulation there is some mild rotation of the vertebrae, usually more in the thoracic than in the lumbar area. As a rule, on the lateral roentgenogram there is some residual anterior angulation in the thoracic area, so that when lateral angulation occurs there is some rotation resulting from this two-plane angulation. The normal lumbar lordosis (posterior angulation) can usually be corrected so that in the lumbar area there is frequently little rotation resulting from two-plane angulation. The amount of rotation depends on the relative persistent lordosis present when the lumbar spine is angulated laterally. In the normal, functional, lateral curve, all the segments, especially the top one, are displaced away from the central spinal axis toward the side of the concavity of the curve.

The anteroposterior roentgenogram of a structural lateral curve (scoliosis) shows characteristic changes in the vertebrae. There is usually some lateral wedging. Any lateral wedging is always a sign of structural change.

The angulation of the vertebrae in the structural lateral curve usually exceeds the functional angulation that is possible in that area. Any significant lateral angulation in the thoracic area, especially in the upper six thoracic segments, is always a sign of structural change. There is usually some abnormal rotation that is greater than the rotation functionally possible in the segments involved, and often there is actual torsion of the vertebrae themselves. The vertebrae in the structural curve are usually displaced from the central axis to the side of the convexity of the curve, whereas the vertebrae in a functional curve are always displaced to the side of the concavity.

Thus, the cardinal roentgenographic signs of a structural lateral curve are

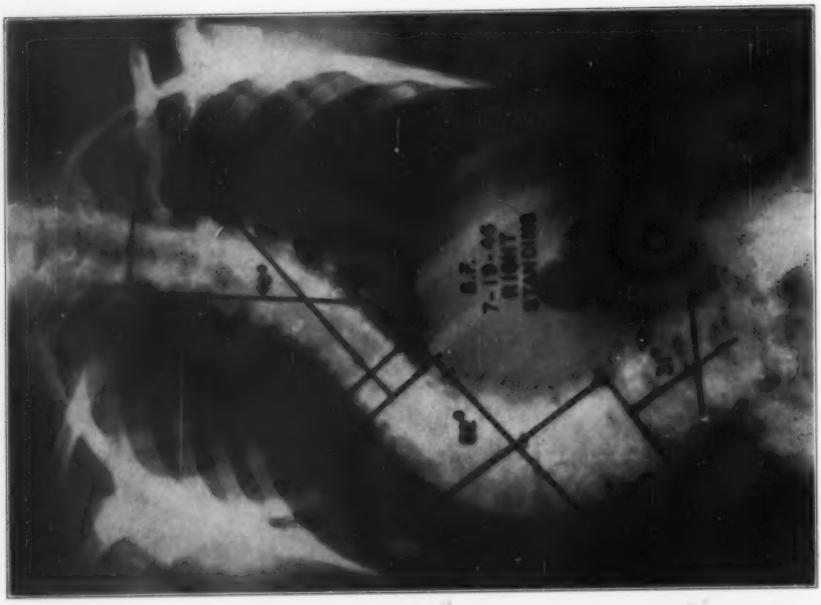

FIG. 2-B

FIG. 2-A

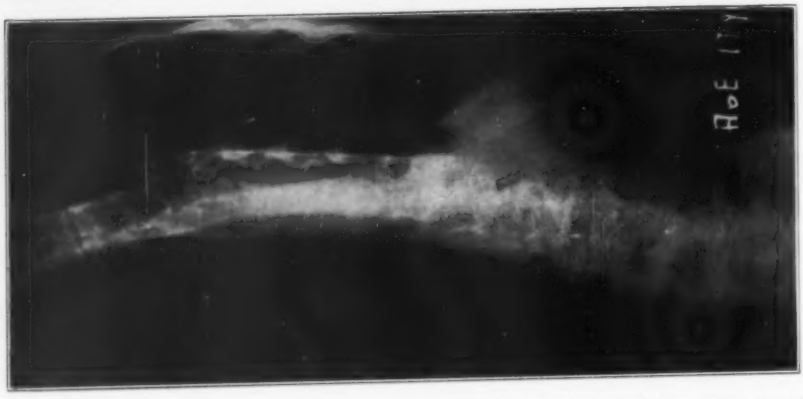

FIG. 1-E

Fig. 1-E: E. F. at seventeen years of age (1954). There is no change in the lower functional curve or fusion area.

Fig. 2-A: S. F. Structural signs in both curves (abnormal wedging, angulation, rotation, and position of vertebrae).

Fig. 2-B: Several years after an orthopaedist fused thoracic curve, thinking it was the primary curve since it did not correct as much as the lower one on bending.

FIG. 2-C

FIG. 2-D

Fig. 2-C: After balancing of the curves and fusion of the lower of two structural curves.
Fig. 2-D: Before and after fusion of the lower structural curve.

abnormal wedging, angulation, rotation, and position of the vertebrae; any combination of these signs may be present.

EXAMPLES OF MULTIPLE-CURVE PROBLEMS

Roentgenograms of several patients with multiple curves illustrate some of the principles just described.

Figure 1-A is the roentgenogram of a girl (E. F.), seven years old, with three mild curves. Even though these are still mild curves, there is evidence of structural changes in the middle thoracolumbar curve. Although this curve is mild and the degree of angulation is within normal limits for a girl this age, the vertebrae in this curve show definite wedging and rotation and their position is abnormal. (If this were a normal functional curve all the segments above the bottom one would be displaced to the left of the bottom segment and to the left of the central spinal axis.) Thus, in this mild curve we see three important signs of structural change: abnormal wedging, rotation, and position of vertebral segments.

On the other hand, we see few structural changes in the lower (lumbar) curve. The angulation, rotation, and position of the vertebrae in this curve are compatible with a functional curve and there is no appreciable wedging of the

vertebrae in this curve. There are, therefore, few signs of significant structural change in this lower curve or, stated another way, there are few indications that there is much abnormal in this curve and a number of signs that it is a functional or secondary curve.

This patient (E. F.) did not have so-called observational therapy. She went to an orthopaedic surgeon who believed in treating scoliosis to prevent an increase. She was treated with braces, plaster casts, and exercises for two years. She and her family believed that she was not improving and when I saw her two years later, the roentgenogram of her spine showed some changes (Fig. 1-B).

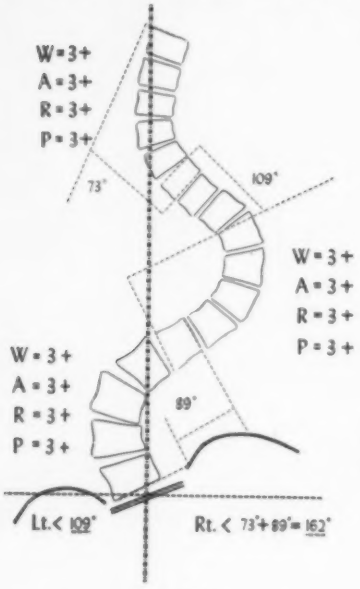

FIG. 3-A

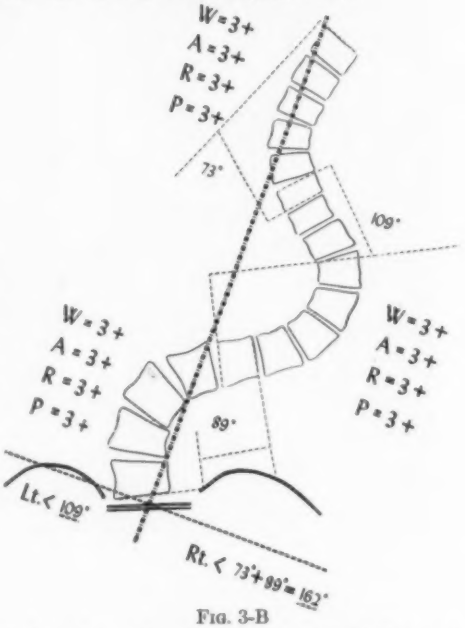

FIG. 3-B

Fig. 3-A: H. B. Three structural curves. All show abnormal wedging, angulation, rotation, and position of vertebrae. (Note pelvic tilt to left.)
Fig. 3-B: Tracing of Fig. 3-A with the pelvis level. (Note the displacement of the head to right.)

Some orthopaedists would say that she had not had the right type of cast, the proper brace, or the correct exercise. I do not believe that any cast, brace, or exercises could have prevented this increase, yet if the curve had not increased, some treatment would have been given the credit.

The middle curve shows vertebral wedging, severe angulation (abnormal for this area), marked rotation (also abnormal for this area), and abnormal position of the vertebral segments—all being displaced from the central axis to the side of the convexity of the curve (not to the side of the concavity of the curve as we see in a functional curve). Thus, there are severe changes in wedging, angulation, rotation, and position of the vertebrae—the four cardinal signs of a structural curve. With this roentgenogram most orthopaedists would have no trouble deciding which is the primary curve or the major structural curve. For many this decision would not be so easy on the basis of the first roentgenogram, made two years before, although there were definite signs of structural changes at that time.

On further study of Fig. 1-B we see that although the lower curve has also increased, the angulation is still within functional limits for this area in a patient

this age (nine years). This lower curve shows very few abnormalities to suggest structural changes. There is little, if any, wedging and the angulation, rotation, and position of the segments are within normal limits.

The upper curve in this curve pattern does not usually tend to increase. Therefore, with such a severe structural (primary) middle curve and no significant structural (primary) changes in the lower curve—which has more characteristics of a functional (secondary) curve—it seemed reasonable to reduce the major structural middle curve to balance with any unreducible structural residuals in the upper and lower curves. This was done without concern about the possible development of structural (primary) changes in the important lower curve. Figure 1-C shows the spine over a year after arthrodesis and Figure 1-E in 1954, about six years after fusion. It is interesting to note that, as had been expected, there was no subsequent problem in the curves above or below the fusion area.

Figure 2-A (S. F.) shows a different problem, with several mild curves in a ten-year-old child. The wedging, angulation, rotation, and position of the segments in the thoracic area suggest definite structural changes in this curve. But there are also abnormalities of wedging, angulation, rotation, and position of the vertebrae in the lower thoracolumbar curve. These changes suggest that we are dealing with two major structural or primary curves. If they are both the result of the same disease or force, then if one curve increased, the other would also be expected to increase. But the orthopaedist who treated this patient obtained bending roentgenograms to decide "which is the primary curve". Then, since the right thoracic convexity did not reduce as much on bending to the right as the left thoracolumbar curve reduced on bending to the left, he concluded that the right thoracic convexity (curve) was the primary curve and did an arthrodesis of this curve. This orthopaedist forgot that there is normally more lateral flexibility in the lumbar than in the thoracic spine and that one should expect the more flexible lumbar curve to be capable of being reduced more than the less flexible thoracic curve.

This patient was not observed, he was treated. The right thoracic convexity (curve), having been shown to be the primary curve, was fused to prevent an increase. Evidently the fusion did prevent an increase in the thoracic curve, but Figure 2-B shows what happened to the lower curve, which had been considered secondary although it had definite roentgenographic signs of a structural (or primary) curve.

The patient was sent to me. Figures 2-C and 2-D show this spine after reduction of the lower curve to balance the upper residual curve and arthrodesis of the lower curve.

Figure 3-A shows three curves in the spine of a patient (H. B.) over twenty-one years of age. It seems obvious that the middle curve (right thoracic convexity) is the most deforming curve and certainly it is desirable to reduce and fuse a curve of 109 degrees. But is it permissible to do so in this situation?

If we study this single erect anteroposterior roentgenogram we see a number of important changes. There are structural changes in all three curves; all three curves show severe wedging, angulation, rotation, and position changes. In other words, this patient has three severe structural or primary curves. Certainly, it is obvious that the severe 109-degree right convexity (curve) is the greatest of the three, but is it the primary curve and are the other curves above and below secondary curves?

The structural changes in the upper and lower curves suggest that they too are structural or primary curves. Although the upper and lower curves individually cause less angulation than the middle greatest curve, together they cause a greater

(162-degree) total angulation to the right than the 109-degree angulation to the left in the greatest curve (middle). Thus, with the pelvis level (Fig. 3-B) the head would be displaced to the right. To make the head level the pelvis must angulate to the left, increasing the left angular deviation of the greatest curve.

We see then that the middle curve is the greatest, but that the sum of the angular deviations produced by the opposite upper and lower curves causes a right deviation of the head. To establish balance it is necessary for the pelvis

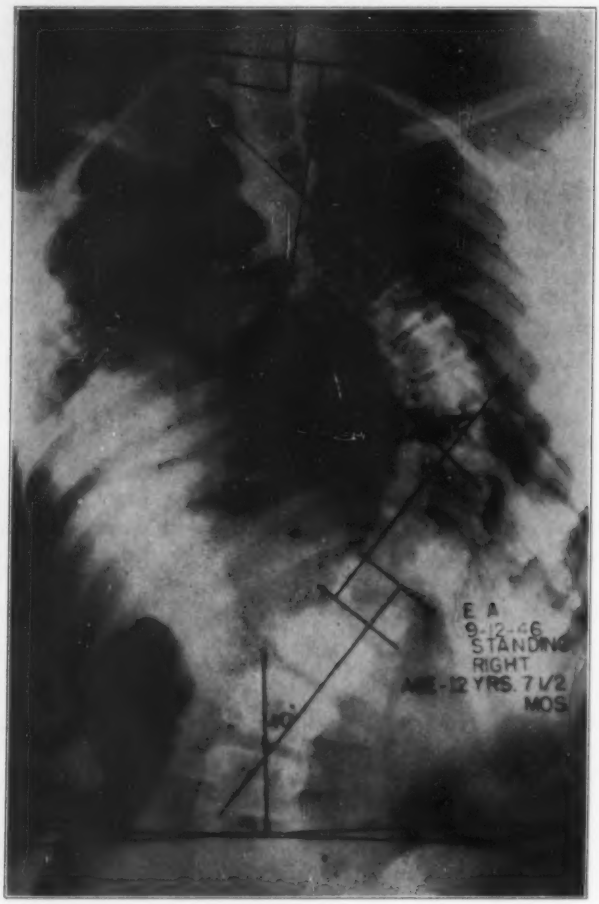

FIG. 4-A

E. A. Three curves, the upper two are structural. The middle one is the major structural curve: abnormal wedging, angulation, rotation, and position of vertebrae.

to angulate to the left, increasing the angulation already produced by the greatest or middle curve. Thus, although it would be desirable to reduce the severe 109-degree thoracic curve, it is not permissible to do so unless one or both of the opposite lesser curves are also reduced to maintain balance.

We might say that in this case there are three major structural curves, and, although the middle one is actually greater than the others, the sum of the angular deviations resulting from the other two smaller curves is really more important and, to get the pelvis level, one or more of the lesser structural curves should be reduced rather than the more obviously greater middle curve. Reduction

of the middle 109-degree curve is desirable but not permissible in this case.

In Figure 4-A (E. A.) there is a different curve pattern, with three curves. The middle curve is sufficiently severe to justify reduction and fusion, but is this permissible? Careful study reveals that there are structural changes in the middle major curve and slight wedging, angulation, rotation, and position changes in the upper and lower curves. This indicates that the upper and lower opposite curves are functional rather than structural and we can predict that the lower curve will reduce well on left bending. It did, and so the major middle structural curve was reduced to balance the opposite curves and fused. Figures 4-B and 4-C show the result after arthrodesis.

FIG. 4-B

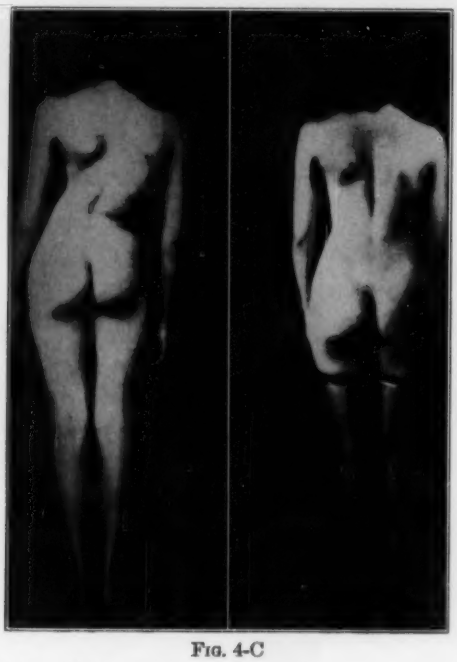

FIG. 4-C

Fig. 4-B: After arthrodesis of the middle major structural curve.
Fig. 4-C: Before and after arthrodesis.

Figure 5-A (M. F.) shows another triple curve with many structural changes in the middle curve and few such changes in the opposite curves above and below. Figure 5-B shows the result after balancing of the curves and fusion of the major structural curve. Figure 5-C shows the patient before and after fusion.

SUMMARY AND CONCLUSIONS

Ferguson's concept of a primary curve was the first important step in the solution of the problem of scoliosis. However, the misuse of the terms *primary curve* and *secondary curve* has caused confusion.

The normal spinal curves in the anteroposterior plane of the erect spine may be used as a basis for defining three types of lateral curves:

1. A structural curve is one with structural changes in bone, nerve, muscle, or other tissues and is a curve which the patient cannot correct and maintain corrected when he is erect;

2. A functional curve is one without structural changes that results from the normal available angulation between vertebrae. It is a curve which the patient can correct and maintain corrected when he is erect;

3. A combined structural-functional curve is one which has some structural and some functional characteristics which the patient can partly correct when erect.

Structural and functional curves have definite characteristics easily noted on a single erect anteroposterior roentgenogram. The cardinal roentgenographic

FIG. 5-A

M. F. Three curves. The middle curve shows abnormal wedging, angulation, rotation, and position of vertebrae. The upper and lower curves show minor structural changes.

signs of a structural curve are abnormal wedging, angulation, rotation, or position of the vertebrae in the curve.

If we remember the characteristics of a normal functional curve and observe the relative amount of abnormal wedging, and angulation, and rotation, and the changes in position of the vertebrae in any curve, it is easy, with a single erect anteroposterior roentgenogram, to determine which curves are major structural curves (primary), which are functional (secondary) curves, and which are combined structural functional or minor structural curves.

Although roentgenograms of the spine bent to either side or sitting with the pelvis tilted are helpful before arthrodesis in deciding how much the opposite

curves will reduce and how much the major curve may be reduced without loss of balance, they are not necessary to determine which curves are major structural (primary) curves.

There are multiple curves in most patients with scoliosis and in view of the confusion caused by the misuse of the terms *primary curve* and *secondary curve* it might be helpful to use the terms *structural curve* and *functional curve.* Since there are very few pure functional curves and most spinal curves have some structural changes we may describe those with major structural changes as major structural curves and those with relatively minor structural changes as minor

FIG. 5-B

FIG. 5-C

Fig. 5-B: Eighteen months after balancing of the curves and arthrodesis of the major middle structural curve.

Fig. 5-C: Before and after arthrodesis.

structural curves. Thus we could describe a common triple-curve pattern as a single major structural curve with minor structural curves above and below, indicating that the middle curve has the greatest structural changes but the curves above and below also have some structural elements. We could describe another problem as double major structural curves with minor structural or functional curves above and below.

In scoliosis it is important to estimate the relative structural changes in the curves and the resultant displacement of the head from its central spinal axis.

NOTE: Anything I have accomplished in orthopaedics I owe to my chief, teacher, and friend, Dr. Philip D. Wilson.

Without the help of my teacher and friend, Dr. Albert Ferguson, I could not have accomplished anything in the problem of scoliosis.

The Effect of a Tibial-Tubercle Transplant in Children on the Growth of the Upper Tibial Epiphysis

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This study concerns the effect of transplantation of the tibial tubercle on the growth of the upper tibial epiphysis when this procedure is performed when the patient is at an early age.

The proximal epiphysis of the tibia appears at birth or shortly thereafter, and the epiphysis of the tibial tubercle becomes apparent when a child is approximately eleven years of age. Normally, these two epiphyses are continuous and they may not fuse until the child reaches the age of nineteen. As is well known, injury to the epiphysis or the tibial tubercle has been observed to cause genu recurvatum. This follow-up study of a consecutive series of young children on whom transplantation of the tibial tubercle had been performed at eleven years of age or younger was undertaken to determine the incidence and type of change in the proximal tibial epiphysis and knee joint produced by this procedure.

CLINICAL MATERIAL

There were sixteen patients in this series, but one was not included because of inadequate follow-up. Of the remaining children, six girls and nine boys, nine had cerebral palsy, and six had lateral dislocation of the patella.

Twenty-four transplantations (nine bilateral and six unilateral procedures) were performed by six surgeons, using the same technique. All twenty-four procedures were done when the children were from seven to eleven years old, and all the children were followed for from two to eleven years—an average follow-up of six years. The ages of these children at follow-up ranged from thirteen to nineteen years, the average being fifteen and a half years. Nine of fifteen children were fifteen years old or older at the time of follow-up.

OPERATIVE PROCEDURE AND SEQUELAE

Surgical Technique

The procedure employed in this series was that described by Bosworth and Thompson (Fig. 1). In this technique, the whole tibial tubercle and its epiphysis are removed as one block and inlayed into a previously prepared bed at a lower and more medial level where the block and the attached patellar tendon are fixed with a three-hole stainless-steel plate and screws. The plate holds the transplant in place, and incidentally serves as a marker for growth studies, since it identifies the region where the transplant was originally placed. The defect at the site from which the tubercle was transplanted is left open to minimize the risk of a bridge of bone forming across the anterior ridge of the epiphyseal plate.

Operative Complications

In three of the patients with cerebral palsy, fractures of the tibia occurred through the defects from which the tubercle had been removed at the time of

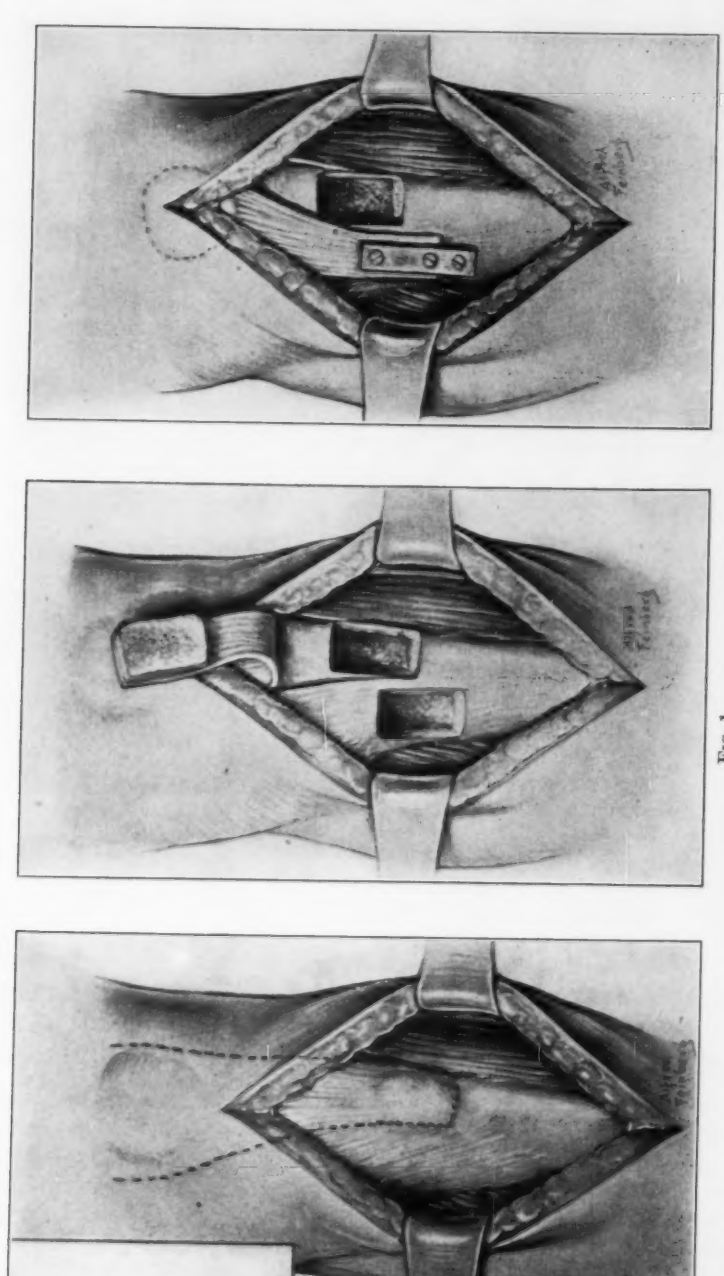

FIG. 1

Surgical technique. Note that the whole tibial tubercle is transplanted and fixed in place with a plate and three screws. (Reprinted from Bosworth, D. M., and Thompson, F. R.: Fixation of the Transplanted Tibial Tubercle. J. Bone and Joint Surg., 28:285–287, Apr. 1946.)

FIG. 2-A

Figs. 2-A through 2-D: The patient was eight years old at the time of operation.

Fig. 2-A: Preoperative roentgenogram.

Fig. 2-B: Roentgenogram showing fracture occurring at the time of surgery. The proximal fragment is angulated.

FIG. 2-B

surgery (Figs. 2-A and 2-B). In one, the fracture was undisplaced, but in the other two, the proximal fragment angulated, causing a posterior angulation at the fracture site. All three fractures united eventually without further treatment. Of the two fractures that angulated, one had persistent deformity, but the upper tibial epiphysis was uninvolved at the time of writing, and further therapy was unnecessary. Restoration of normal alignment occurred in the other one as the result of growth.

Patellar Changes

Because nine of the fifteen patients had bilateral operation, it was not possible to evaluate the changes by comparison with the opposite side. In the six unilateral tibial operations, it appeared that the articular surface of the patella conformed to the contour of the adjacent structures (Figs. 3-A through 3-E). Similar changes were observed in both knees of the patients who had bilateral operations.

At the time of surgery, the patella was advanced from zero to one and one-quarter inches, making an average advancement of five-eighths of an inch as measured from the tibial epiphyseal line to the center of the patella. In six the relative position of the patella remained unchanged with growth; in nine, further distal advancement of as much as one-half of an inch seemed to take place as growth continued; and, in eleven, the patella moved proximally as much as one-half of an inch. It would appear that the degree of patellar advancement

FIG. 2-C

FIG. 2-D

Fig. 2-C: Roentgenogram showing healing and realignment. Note the downward movement of the plate.

Fig. 2-D: Roentgenogram made at eleven-year follow-up, when the patient was nineteen years old.

at operation remains essentially unchanged with growth, and that the posterior contour of the patella tends to mold itself to conform to the structures with which it articulates.

Position of the Plate

In all cases the plate and screws "moved downward" with growth, as would be expected. The greatest amount of displacement was two and one-half inches, as measured from the proximal epiphyseal line of the tibia to the upper limit of the plate; this occurred in a girl with cerebral palsy whose left tibial tubercle was transplanted when she was nine years old, and whose last roentgenogram was made when she was nineteen years old (Figs. 4-A through 4-D). The least "downward" migration, which amounted to one-half of an inch, occurred in a child whose tibial tubercle was transplanted at ten years of age because of lateral dislocation of the patella but this patient was followed for only two years after operation, making this the shortest follow-up in this series.

The amount of "downward" movement of the plate was directly proportional to the amount of growth still remaining in the tibia at the time of operation.

FIG. 3-A FIG. 3-B FIG. 3-C

Figs. 3-A through 3-E: The patient was eight years old at the time of surgery and was sixteen at the time of writing.

Fig. 3-A: Note the flat contour of the articular surface of the patella before operation. Compare with the normal side shown in Fig. 3-D.

Fig. 3-B: The situation immediately after surgery.

Fig. 3-C: Two years after operation. Note that the contour of the posterior aspect of the patella now corresponds to the structures posterior to it, whereas on the normal side (Fig. 3-E) the posterior surface of the patella still has a relatively flat contour.

Roentgenograms at follow-up frequently showed that the plate and screws were submerged by an overgrowth of bone.

Tibial Tubercle

In all cases the epiphysis of the tibial tubercle failed to develop in its transplanted position or from the epiphyseal remnants that might have been left behind at the time of transplantation (Fig. 5). In all these fifteen cases there was consequent failure of the development of the normal bone prominence associated with the tibial tubercle. This produced narrowing of the tibia in this region in the anteroposterior plane, but no other defect.

Patellar Tendon

In all cases the transplant displaced the insertion of the patellar tendon downward and medially, fixing it firmly to the shaft of the tibia and removing the epiphyseal line that normally separates the tibial tubercle from the rest of the tibia. As the proximal tibial epiphysis grew upward, the distance between the upper epiphyseal line of the tibia and the plate and screws used for fixation increased. Since the tendon was originally placed at the level of the plate and screws, we were interested to determine if the tendon remained attached at this fixed point or migrated upward, maintaining a constant distance from the upper epiphyseal line of the tibia. Since the patella itself remained more or less stationary and did not shift upward or downward more than one-half inch with growth, it appeared that either the patellar tendon had lengthened or its attachment had migrated upward. Clinical examination of these knees and soft-tissue roentgenograms at follow-up indicated that upward migration had occurred. In one instance

FIG. 3-D FIG. 3-E

Fig. 3-D: The normal side.
Fig. 3-E: Roentgenogram showing the normal side, made at the same time as Fig. 3-C for comparison.

the plate was removed because of overlying tenderness four years after operation. In this child the plate had "moved downward" one and three-eighths inches, and the patella had remained at essentially the same level as it was at the completion of the operation. The transplanted insertion of the patellar tendon appeared to have migrated proximally, maintaining its normal distance from the knee joint as growth of the tibia displaced the plate "downward".

Proximal Tibial Epiphysis

Transplantation of the tibial tubercle involves complete disruption of the traction epiphysis of the patellar tendon. Since this epiphysis is in extremely close proximity and anatomically continuous with the proximal tibial epiphysis, this surgical procedure might be expected to cause some disturbance of growth of the upper end of the tibia or knee joint. With this in mind, the results of these twenty-four operations were carefully evaluated.

In fourteen patients (twenty-three knees) the growth pattern in the region of the upper tibial epiphysis was normal except for the absence of the tibial tubercle. There was no tendency for arrest of growth of the anterior portion of the upper tibial epiphysis, no tendency for earlier epiphyseal closure, and no varus or valgus deformity.

In the remaining patient, a girl with bilateral dislocation of the patella who was eleven years old at the time of surgery, recurvatum deformity of one knee was first observed five months after operation. Two years after operation this deformity had increased to 32 degrees (Figs. 6-A through 6-C).

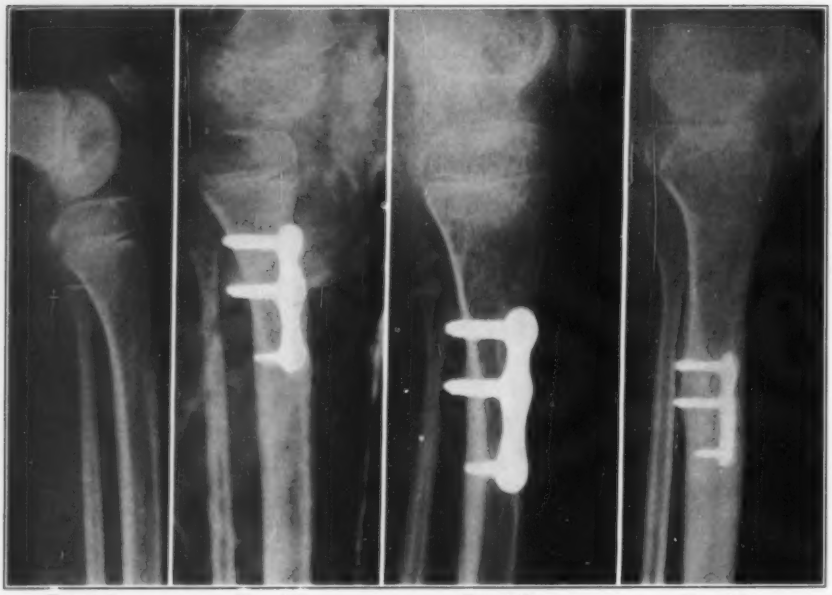

FIG. 4-A FIG. 4-B FIG. 4-C FIG. 4-D

Figs. 4-A through 4-D: Roentgenograms showing the "downward movement" of the plate and screws over a period of ten years.
Fig. 4-A: Preoperative roentgenogram, made when the child was nine years old.
Fig. 4-D: Roentgenogram made when the patient was nineteen years old.

When the child was twelve years old, a corrective osteotomy was performed in the proximal part of the tibia at the level of the deformity. This patient has now been followed for six years since operation. When she was last seen at the age of eighteen there was no recurrence of the deformity. The cause of the genu recurvatum in this patient is not clear. However, the postoperative roentgenogram (Fig. 6-B) shows a small portion of the epiphysis of the tibial tubercle was left projecting downward, in contrast to the other cases in which this epiphysis appeared to have been removed completely up to and beyond the level of the proximal tibial epiphyseal plate. It is possible that this projecting fragment resulted in the development of a bone bridge and the arrest of growth of the anterior portion of the epiphyseal plate similar to the partial growth arrests produced experimentally by Friedenberg. This occurrence of this one deformity emphasizes that damage to the proximal tibial epiphysis may occur and that, if this procedure is used, care should be taken not to injure, in any way, the proximal epiphyseal plate.

The proximal fibular epiphysis was also considered in this study in order to determine if any discrepancy in the relative lengths of the tibia and fibula had developed as the result of stimulation of the proximal tibial epiphysis by the trauma of surgery. In only one instance did it appear that a spurt of growth in the proximal tibial epiphysis had produced a discrepancy. In this child, who was eight years old at the time of operation, the tibial epiphyseal plate was three-eighths of an inch higher than that of the fibula eight years after operation when the patient was sixteen years old.

SUMMARY AND CONCLUSIONS

Distal and medial transplantation of the tibial tubercle was performed twenty-four times in fifteen children who were in their eleventh year or younger.

Fig. 5: Roentgenogram four years after operation, showing the failure of development of the tibial tubercle. The soft-tissue shadow, representing the patellar tendon, is in an essentially normal location. The plate and screws have "moved downward".

Figs. 6-A through 6-C: Roentgenograms of a girl who had transplantation of the tibial tubercle for lateral dislocation of the patella.

Fig. 6-A: Lateral dislocation of the patella.

Fig. 6-B: Postoperative roentgenogram made after distal and medial transplantation of the tibial tubercle. Note the small beak of the tibial epiphysis projecting downward.

Fig. 6-C: Two years after transplantation of the tibial tubercle, the proximal tibial epiphyseal plate has closed, and a recurvatum deformity of 32 degrees has developed.

FIG. 5

FIG. 6-A FIG. 6-B FIG. 6-C

A follow-up study two to eleven years after operation revealed the following data:

The distally displaced patella, with but minor variations, tended to remain in the same position relative to the knee joint as growth continued; the articular surface of the transplanted patella showed some evidence of being remodeled to conform to the shape of the articular surfaces of the underlying femur and tibia;

The tibial tubercle failed to develop in either the normal or transplanted positions;

The attachment of the patellar tendon remained in its normal position as growth continued, migrating upward as the tibia increased in length; there was no evidence that the patellar tendon lengthened, except to the extent expected with normal growth;

With growth in length of the tibia, the epiphysis and metaphysis moved away from the plate and screws that had been used to fix the transplanted tibial tubercle. As a result, the plate and screws were found to be located on the diaphysis at follow-up;

With the technique employed, disturbance of growth of the proximal end of the tibia occurred in only one case—that in which a recurvatum deformity of sufficient severity developed to require corrective osteotomy;

The growth and development of the proximal tibial epiphysis and knee joint were not affected in any of the patients in these cases.

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A Study of the Residual Nitrogen in Bone Following Extraction with Ethylenediamine*

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The observation by Williams and Irvine that ethylenediamine, when used as a solvent in a soxhlet extractor, would remove the organic matrix of bone provided the basis for an interesting concept in bone grafting. It was reasoned that removal of the organic matrix might eliminate the fractions of the matrix that are associated with the undesirable aspects of either the heterograft or homograft response. The basic method of Williams was first used for the preparation of bone grafts by Losee and Hurley, who initially used the term, anorganic bone. However, it was subsequently found that the final product extracted with ethylenediamine was characterized by a substantial decrease in the organic component but not complete removal of the fraction [5]. These investigators further noted that the final extracted bone retained significant amounts of citrate and a trace of nitrogen. However, the exact concentration of the residual nitrogen has remained somewhat controversial, for observers have reported a variation of from 0 to 0.040 per cent, using essentially the same method [1,4].

When ethylenediamine-extracted bovine bone was implanted in substantial defects in the radii of adult mongrel dogs, it evoked an adverse histological response characterized by the encapsulation of the graft by the host and a markedly decreased rate of revascularization and resorption. This foreign-body response was considered most unsatisfactory when compared with the host response to fresh viable autografts, homografts, and even heterografts implanted in a similar way [3].

In an effort to determine the biochemical characteristics of the residual nitrogen in ethylenediamine-extracted bone, the following investigations were attempted: (1) the establishment of the range of concentration of the residual nitrogen in ethylenediamine-extracted bone, (2) the determination of whether any or all of the residual nitrogen represented protein or protein fragments, and (3) the evaluation of this method as a practical laboratory procedure for the extraction of nitrogen in the preparation of nitrogen-free bone**.

MATERIAL AND METHODS

In order to obtain controls for blank determinations and a proper vehicle for studies of the recovery of nitrogen, a large sample of ethylenediamine-extracted bone was finely ground, and a weighed specimen was placed in a muffle furnace at 600 degrees centigrade for one hour. After cooling, the specimen was again weighed, and the percentage of weight loss was calculated. Aliquots of the heated bone that were used as blanks and as recovery vehicles were calculated on the basis of preheated weights. A Kjeldahl determination for nitrogen on the heated

* The opinions and views expressed are those of the authors and are not to be construed as official or necessarily reflecting those of the Medical Department of the United States Navy or the Naval Service at large.

** The amount of residual nitrogen in the extracted bone in grams of nitrogen per 100 grams of extracted bone is expressed as per cent.

material was negative. It was assumed that this nitrogen-free bone would closely approximate the salt effect exhibited by the ethylenediamine-extracted bone in the analyses, and this material was used as a vehicle to which standards were added for studies of the recovery of nitrogen.

The nitrogen content of the ethylenediamine-extracted bone was determined with a Kemmerer-Hallett distillation apparatus after digestion of the bone in fifty-milliliter Kjeldahl flasks for three hours, using a copper catalyst. The size of the samples of ethylenediamine-extracted bone was 500 to 750 milligrams. Fifteen milliliters of the distillate from the Kjeldahl flask was collected in ten milliliters of 0.5 per cent sulphuric acid, and the color was developed by the addition of five milliliters of Nessler's solution. The validity of the method under the experimental conditions was established by the use of the heated, nitrogen-free bone to which a known concentration of ammonium sulphate or serum protein was added. A calibration curve was prepared in a similar manner by the determination of the nitrogen content after the addition of aliquots of a standard ammonium-sulphate solution to the heated bone. Each determination of nitrogen was carried out on a series of four specimens consisting of a blank, a standard, and duplicate samples of the ethylenediamine-extracted bone being analyzed.

Extraction of the whole bone was carried out in a soxhlet apparatus with ethylenediamine at the rate of approximately three cycles per hour. The standard time of extraction was fifty hours. In two of the experiments, small chunks of bovine bone were placed in a sintered-glass thimble, and specimens were removed after fifty, seventy-five, and 105 hours of extraction.

Tap water was found to be unacceptable as a washing medium for the extracted bone because the nitrogen content of the washed bone was found to be relatively high when tap water was used. The following procedure was found to be satisfactory for the removal of excess solvent after extraction.

After removal from the soxhlet apparatus, the bone was placed in a one-liter beaker of distilled water. The beaker was then swirled slowly while the water was changed every ten minutes during the first hour. Thereafter, the water was changed fifteen times during the next two days. The bone was then transferred to a sintered-glass filter of medium porosity and washed with a slow-running flow of distilled water for a period of three days. The final wash solution was slightly acid and gave a negative ninhydrin reaction.

Twenty-three specimens of cortical bone were used to establish the range of the concentration of residual nitrogen in the ethylenediamine-extracted bone. These specimens included eight groups of mice femora, six human bones, and nine bovine specimens. The bones were scraped free of soft tissue, and the human and bovine specimens were sawed into thin strips for extraction. This experiment was also designed to determine if the species of origin of the bone being extracted had any influence on the concentration of the residual nitrogen found after extraction. The influence of the density of the bone and of the permeability of the bone to the solvent on the efficiency of extraction was studied by including three specimens of cancellous bovine bone in the experimental material. It was anticipated that the eight groups of mice femora, which were thin translucent shells of bone after extraction, would yield information regarding the effects of bone density.

For the determination of the amino acids in ethylenediamine-extracted bone, the bone was hydrolyzed, and the hydrolysate was analyzed for amino acids. To test the method, small pieces of washed collagen film* were added to samples of heated bone for recovery studies. Acid hydrolysis was carried out with eight normal (8 N) sulphuric acid for eight hours at 100 degrees centigrade. Sample

* Obtained from The American Cyanamid Company.

sizes ranged from fifteen to twenty-five grams. The ratio of acid to bone was five milliliters per gram. After hydrolysis, the material was filtered through a sintered-glass filter of medium porosity and washed with small portions of distilled water. Kjeldahl determinations on the residue and hydrolysate demonstrated that 95 to 100 per cent of the nitrogen was in the hydrolysate. Basic hydrolysis was carried out using barium hydroxide. The weight ratio of base to bone was 2.5 to 15. The hydroxide and bone were ground together in a mortar and pestle. The mixture was transferred to a large Petri dish and enough distilled water added to form a thin paste. The material was then placed in an oven at 100 degrees centigrade for three hours. The dry residue was removed and extracted with distilled water followed by centrifuging. The extraction procedure was repeated four times. The washings were combined and neutralized to pH 7 with 10 per cent sulphuric acid. The neutral solution was separated by centrifuging and evaporated to a final volume of 0.5 to one milliliter in an oven at 100 degrees centigrade. The nitrogen in the final solution after basic hydrolysis was determined by the Kjeldahl method to establish what percentage of the nitrogen present in the ethylenediamine-extracted bone before basic hydrolysis was transferred to the hydrolysate and washings.

The method of Van Slyke for the determination of amino-acid carbon dioxide was used to estimate the amino-acid nitrogen present in the acid hydrolysates. A pH of 2 to 3 (indicator paper) was obtained by treating the samples with five normal (5 N) sodium hydroxide. At this acidity, the heavy precipitate of calcium salts was avoided; all the specimens showed only a faint turbidity. The recovery samples that contained the collagen film were analyzed in the same manner as the unknown samples. Positive artefacts were ruled out by a determination on duplicate specimens without the addition of ninhydrin.

The method of Levy and Chung was used for the separation of the amino acids by two-dimensional paper chromatography. The solvent systems were butanol, acetic acid, and water for the initial run, and phenol, cresol, and borate buffer for the second run. The neutral concentrate from the alkaline hydrolysis was used for this study. Acid hydrolysis gave fair results, but, owing to the high salt concentration, even in those hydrolysates neutralized with barium hydroxide, the resolution was poor.

RESULTS

The experiments designed to establish the validity of the Kjeldahl nitrogen determinations under the experimental conditions demonstrated a recovery of 97 to 101 per cent with both protein and ammonium-sulphate standards. The potential source of error in the analytical studies of the residual nitrogen by the Kjeldahl analysis was not considered significant.

The results of the study, in which time of extraction and type of bone tested were the variables, are shown in Table I. The average residual nitrogen concentration in the twenty-three specimens of cortical bone after fifty hours of extraction with ethylenediamine was 0.0145 per cent. The observed range was 0.0109 to 0.0173 per cent.

The Van Slyke method for the determination of amino-acid carbon dioxide yielded a recovery of 73 per cent of the theoretical nitrogen content in the experiment using collagen film as a control. Therefore, the correction factor of $\frac{10}{7.3}$ was applied to the results of the analysis of the ethylenediamine-extracted bone. An approximation of the amino-acid nitrogen in four of the specimens of ethylenediamine-extracted cortical bone determined by this method of calculation indi-

TABLE I

RESIDUAL NITROGEN CONCENTRATION OF EXTRACTED BONE

Type of Bone	Species of Bone	Number of Experiments	Time of Extraction (*Hours*)	Average Nitrogen Concentration (*Per cent*)
Cortical	Mice	8	50	0.0138
	Men	6		0.0147
	Cattle	9		0.0149
Cancellous	Cattle	3	50	0.0093
Cortical	Cattle	2	50	0.0141
		2	75	0.0122
		2	105	0.0117

TABLE II

AMINO-ACID ANALYSIS OF ALKALINE HYDROLYSATE

Amino Acid	Micromoles per 0.1 Milliliters	Amino Acid	Micromoles per 0.1 Milliliters
Methionine sulphoxide	0.010	Valine	0.057
Hydroxyproline	0.057	Methionine	0.010
Aspartic acid	0.106	Isoleucine	0.029
Threonine	0.038	Leucine	0.241
Serine	0.089	Tyrosine	0.029
Glutamic acid	0.325	Phenylalanine	0.105
Proline	0.162	Lysine	0.123
Glycine	0.491	Histidine	0.029
Alanine	0.332	Arginine	0.029
		Ammonia	0.057

cated that 42 to 50 per cent of the total nitrogen in the ethylenediamine-extracted bone was present as amino acids. The concentration of amino-acid nitrogen was of necessity an approximation, since the modification of the method for an acid hydrolysate of this type resulted in questionable accuracy.

The qualitative studies by chromatographic separation yielded a spectrum of amino acids as anticipated. Fifteen spots were isolated with questionable detection of proline. We did not observe a spot corresponding to hydroxyproline in the hydrolysate from the ethylenediamine-extracted bone. The hydrolysate from the collagen film yielded a spot with an R_f very similar to one obtained with a hydroxyproline standard. However, when this spot was sprayed with ninhydrin, this color was much duller than the bright cherry-red color obtained with the standard.

One ten-gram sample of ethylenediamine-extracted cancellous bone containing 0.0091 per cent of nitrogen was hydrolyzed by the alkaline method and analyzed on an automatic amino-acid analyzer similar to the one described by Spackman, Stein, and Moore. This specimen was chosen for this type of analysis because of its low nitrogen concentration, which was considered to indicate nearly complete extraction of protein. The results of the amino-acid determinations with the analyzer are shown in Table II. From these results it was calculated that 39 per cent of the nitrogen in the original ten-gram sample of cancellous bone could be accounted for as amino-acid nitrogen. However, this figure had no quantitative significance, since the Kjeldahl determinations of the nitrogen content of the basic hydrolysate and washings of the ethylenediamine-

extracted bone demonstrated that only 53 per cent of the nitrogen, originally present in the ethylenediamine-extracted bone, was transferred to the final solution. Repeated washings of the hydrolyzed material failed to increase the yield of nitrogen. The spectrum of amino acids found does not permit us to predict the presence of a homogenous protein residue. The presence of hydroxyproline indicates that some collagen was present in the extracted bone. However, the absence of hydroxylysine, as well as the very high ratio of other amino acids such as glycine and glutamic acid to hydroxyproline, indicates that collagen can account for only a small amount of the amino-acid nitrogen present. The loss of cystine and cysteine, as well as the deamination that occurs during alkaline hydrolysis, must be considered in addition to losses caused by adsorption on the abundant precipitate that forms during neutralization of the hydrolysate. Considering the qualitative significance of the amino acids rather than their relative concentrations, which are of questionable significance, we would speculate that the amino acids, not accounted for, represent a residue of one of the minor components of whole bone, such as the mucoprotein or "resistant protein" [2], which is not extracted by ethylenediamine because of binding forces within the crystal structure.

Alkaline hydrolysis and separation by paper chromatography were applied to a sample of the ethylenediamine-extracted bone obtained commercially *, with results similar to those observed with samples extracted in our laboratory.

DISCUSSION

From the results in these experiments using bone of different types and of different species of origin, as well as bone extracted for different periods of time, it would appear that the residual nitrogen is not a predictable function of the extraction procedure. Extraction for 105 hours did not yield a nitrogen concentration significantly different from that obtained by seventy-five hours of extraction when due allowance is made for the increased time. The results observed with cancellous bone indicated that a more efficient extraction could be obtained by increasing the porosity or decreasing the particle size of the specimens, but this observation was not confirmed by the results in the experiments with mice femora (Table I). It appears probable that the concentration of residual nitrogen in the ethylenediamine-extracted bone is related to the concentration of one or more of the organic components present in the whole bone prior to extraction.

In ethylenediamine-extracted bone, we have been unable to identify qualitatively any material that would account for the observed difference between the total residual nitrogen and the apparent concentration of amino-acid nitrogen. Our results indicate that this unknown material accounts for 50 to 60 per cent of the nitrogen content. An experiment was designed to ascertain if chunks of heated ethylenediamine-extracted bone, which contained only traces of residual nitrogen, were capable of binding ethylenediamine when subjected to a second extraction for fifty hours in the soxhlet apparatus. The results of this experiment demonstrated no difference in the results of the Kjeldahl determinations before and after the second extraction. However, before attaching any significance to the findings in this experiment, subtle changes in the crystal structure of hydroxyapatite, the result of exposure to elevated temperature, must be considered. Such alterations in the crystal structure might possibly affect the binding capacity of bone for the ethylenediamine solvent. However, the results may be considered as an indication that the ions of the bone crystals will not form complexes and thus retain the solvent.

* Armour and Company.

The high concentration in tap water of metallic ions that are capable of forming complexes with ethylenediamine may account for the high nitrogen content of bone washed with this medium. The absorption of the metallic complex on the bone crystal would yield an elevated nitrogen concentration when the bone was subjected to Kjeldahl analysis. This situation was not observed when distilled water was used throughout the washing procedure.

We were unable to confirm the observation that nitrogen-free bone was prepared by extraction with ethylenediamine [1]. Our results indicated that a negative nitrogen concentration is impossible to obtain with this solvent. The biological significance of the residual nitrogen in the ethylenediamine-extracted bone is as yet undetermined. It is possible that residual protein and other ill-defined organic materials that have been subjected to high temperatures and to the solvent action of ethylenediamine may be cytotoxic. Therefore, we believe that the host response to anorganic or ethylenediamine-extracted-bone grafts cannot be considered as characteristic of the host response to grafts of so-called pure bone crystals.

SUMMARY AND CONCLUSIONS

The range of concentration of residual nitrogen in twenty-three specimens of cortical bone that had been extracted with ethylenediamine was found to be 0.0109 to 0.0173 per cent.

The concentration of nitrogen remaining in bone after fifty hours of extraction with ethylenediamine was not greatly reduced by more prolonged extraction; the concentration of the nitrogen does not appear to be a predictable function of extraction after fifty hours.

There was no evidence to indicate that nitrogen-free bone might be prepared by extraction with ethylenediamine without modifying the solvent.

Analyses of the acid hydrolysates of four specimens of ethylenediamine-extracted cortical bone by the Van Slyke method for the determination of amino-acid carbon dioxide indicated that 42 to 50 per cent of the residual nitrogen was present as amino acids. An analysis of the alkaline hydrolysate of one cancellous specimen of the ethylenediamine-extracted bone by an automatic amino-acid analyzer indicated that 39 per cent of the residual nitrogen was present as amino acids. The results of these two methods of analysis indicate that 40 to 50 per cent of the residual nitrogen present in bone after extraction with ethylenediamine is present as protein or protein fragments.

Note: The authors wish to acknowledge the excellent technical assistance of Chief Hospital Corpsman H. M. McClintic, United States Navy, during the course of this work and to express their gratitude to Dr. K. A. Piez, of the Biochemistry Laboratory, National Institute of Dental Research, for the separation of the amino acids on the automatic analyzer.

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Proceedings

THE AMERICAN ORTHOPAEDIC ASSOCIATION

ANNUAL MEETING: THE HOMESTEAD, HOT SPRINGS, VIRGINIA
MAY 30 THROUGH JUNE 2, 1960

Myelodysplasia. DR. LEONARD F. BUSH [1] discussed various types of congenital anomalies in the lumbar and thoracic areas. He reported on a series of nineteen patients having a variety of deformities: seven with diastematomyelia; seven with filum terminale syndrome; two with typical myelomeningocele; and three with epidural or perineural cysts. Fourteen myelograms and twelve operations were performed.

Case reports were presented, stressing the application of orthopaedic surgery in these deformities. Progressive deformities of the lower extremities accompanied by symptoms of bladder or bowel difficulty should suggest further study and may be relieved by surgery. Surgery aimed at allaying progression of the deforming lesion was reviewed. Three epidural or perineural cysts found in the upper sacral area produced typical sciatic pain.

Spondylolisthesis. Etiology and Comparative Efficacy of Various Surgical Procedures. DR. L. L. WILTSE [2] reported on consanguinity studies of thirty-four families, in each of which there was a person with spondylolisthesis originally seen by him in office practice. In these families, 30 per cent of each patient's relatives who were examined had a defective pars interarticularis, in comparison with an incidence of 5 per cent in the general American population. Only direct forebears, direct descendants, and siblings were included in the statistics; the original patient was never included because he was selected.

Dr. Wiltse believed that the defective pars interarticularis is due to two factors:

1. A hereditary dysplasia of the arch of the affected vertebra and, probably, of several vertebrae; this dysplasia seems to be characterized by a lack of the usual ability of bone to repair itself;

2. The particular stresses on the lower lumbar spine that are present only in man because of his erect stance and lumbar curve.

The fifth lumbar vertebra is usually involved because it is subjected to the most stress, but there are many instances of involvement of other vertebrae, and even of involvement of several vertebrae in the same person.

Dr. Wiltse presented roentgenograms of identical twins. The twenty-fourth segment was involved in one twin, and the twenty-fifth in the other. He believed that this finding indicates dysplasia of several vertebral arches, not necessarily only the involved vertebra in a given person.

The defect is never present at birth, it is almost always present by the sixth year of life, and it is certainly always present by the eighteenth year of life, if it is going to occur at all. If any slip is going to occur, it occurs in the first few years after the appearance of the lesion, except in rare instances.

The results of three methods of surgical treatment for spondylolisthesis were compared; these were fifteen posterior-element fusions, twenty-five posterior elements removed without fusion, and eight interbody fusions.

Posterior-element fusion was considered the best method of treatment despite a 20 per cent incidence of failure of the first operative attempt. Posterior-element removal, when done alone, resulted in at least some further slipping in 60 per cent of the cases. In one patient, progressive forward slipping did not start until more than a year after operation, and, when last checked, had slipped 28 per cent. Interbody fusion failed to produce fusion in every case.

1. Geisinger Memorial Hospital, Danville, Pennsylvania.
2. 909 Pine Avenue, Long Beach 13, California.

Spondylolisthesis and Tight Hamstrings. Dr. George S. Phalen [3] and Dr. James A. Dickson [4] discussed the association and possible causal relationship of advanced degrees of spondylolisthesis and tight hamstring muscles, the latter being so marked as to cause a disfiguring limp. Tightness of the hamstrings may be the only abnormal physical finding associated with irritation of the cauda equina from any cause. Pain was not a prominent feature of this syndrome.

The authors said that instead of stretching or lengthening of the hamstring tendons, the proper treatment was decompression of the cauda equina by removal of the loose lamina, followed by spine fusion.

Surgical Treatment of Spondylolisthesis. Dr. Arthur A. Thibodeau [5] reviewed the extensive literature on spondylolisthesis and reported a study of 244 cases observed over ten years. Of these, approximately 30 per cent had no symptoms, and only 21 per cent required surgical treatment. In seventy, the diagnosis was made incidentally. Emphasis was placed on the role of degenerative changes in the causation of symptoms.

The surgical procedures employed by the author were described. In the few cases in which simple decompression had been performed (the Gill operation) the results had been disappointing. It had been necessary to do interbody fusion in a few instances, using an extraperitoneal or transperitoneal approach, but this formidable procedure was believed to be seldom indicated. If symptoms of nerve-root irritation were present, removal of the loose laminal arch and decompression of the nerve roots were recommended. The author stated that a solid fusion bridging the defective vertebra will give complete and permanent results in the majority of patients. He favored a method of fusion in which long cortical grafts were inserted into lateral grooves on each side, so placed that they join the fourth and fifth lumbar vertebrae to the sacrum. These two grafts were also in contact with an overlying H-graft inserted after the manner of Bosworth. In thirty-seven patients treated by this method there were only two with pseudarthroses, and they did not have disabling symptoms.

Bone-Density Evaluation in Relation to Orthopaedic Management. Dr. C. F. Fercıot [6] described a method of measuring the relative density of bone structure, using aluminum step wedges (in .015-inch gradations from .060 to .150 inch in thickness) that were placed on the film at the same time the roentgenogram of the patient's hand was being made. This has the theoretical advantage that variation in roentgenographic and film-processing techniques affects the standard in the same way that it affects the test object, which in his series was the proximal phalanx of the long finger.

Measurements were made with an Eastman color densitometer, and the bone densities were recorded in thousandths of an inch of aluminum. One hundred and fifty determinations were made on patients chosen at random. The film exposures and development were standardized as much as possible. Those persons in whom the author found low bone density seemed to fall into a typical category, mostly women who were past menopause and women who had had hysterectomies. He also noted that bone density decreased with age and after the prolonged use of corticosteroids. Low bone density was also demonstrated in patients who had had compression fractures of the spine with minor trauma and in patients with Paget's disease or multiple myeloma. He noted that in a group of patients with hip prostheses, those with a normal bone density had better results than those with a low bone density; in some patients with a low bone density there was loosening of the prosthesis. In several of these patients, a satisfactory clinical improvement as well as improvement in bone density was obtained after "anabolic" medication.

The author concluded that bone density measured in the hand was a good reflection of the general skeletal condition and a useful guide in clinical practice.

Aneurysmal Bone Cyst. Dr. William F. Donaldson, Jr.[7], stated that he was convinced that aneurysmal bone cyst represents a distinct clinical, pathological, and roentgenographic entity. He believed that it was not a secondary lesion produced by some unknown change in a pre-existing lesion. Trauma did not appear to be a factor in the production of the lesion, but seemed to be, in most instances, the exciting factor that caused the patient to seek medical aid.

He agreed with Lichtenstein in the opinion that the etiology was probably some aberrant hemodynamic vascular change.

Six examples were shown.

3. 2020 East 93rd Street, Cleveland 6, Ohio.
4. 10515 Carnegie Avenue, Cleveland 6, Ohio.
5. New England Medical Center, 37 Bennet Street, Boston 11, Massachusetts.
6. 1000 South 13th Street, Lincoln 8, Nebraska.
7. 121 University Place, Pittsburgh 13, Pennsylvania.

Dr. Donaldson said that aneurysmal bone cyst is a benign lesion, which, when it occurs in the long bones, is typically seen in the metaphyseal area and is usually eccentrically located. It has an expansile soap-bubble character that extends beyond the normal confines of the bone. It is outlined by subperiosteal new bone. When it occurs in the spine, it involves chiefly the posterior elements and is characterized by enlargement and distention of their outlines. Pathologically, it is an expansile lesion delineated by subperiosteal new bone, containing a pulpy red tissue that bleeds freely and contains blood spaces. Microscopically, it consists of a lining and septum of active fibrous tissue containing osteoid, blood spaces, and foreign-body giant cells. The blood spaces contain erythrocytes of normal appearance. The linings of these blood spaces are, for the most part, not covered by endothelium, and the walls do not have muscular coats.

The diagnostic importance of the histology of the septa was stressed.

Two of three cases of involvement of a posterior element in the spine showed the clinical picture of paraplegia.

Surgical curettement and decompression result in complete recovery of neurological deficit.

He recommended surgical treatment. Curettement with or without bone-grafting was performed wherever possible. Roentgenographic therapy was beneficial when excision was incomplete or the lesion inaccessible.

Pigmented Villonodular Synovitis with Invasion of Bone. Report of Six Cases. Dr. Paul E. McMaster [8] (J. Bone and Joint Surg., **42-A:** 1170–1183, Oct. 1960).

Eosinophilic Granuloma. "One of Three Sisters". Dr. Charles R. Rountree [9] and Captain Charles A. Rockwood, Jr.[10], reported on seven new cases of eosinophilic granuloma. Their review of the literature included the description, in 1929, of a myeloma of the frontal bone. In 1940 Otani and Ehrlich referred to this disease as a solitary granuloma of bone; at the same time Jaffe and Lichtenstein called it eosinophilic granuloma, and, in the next few years, they and other workers showed that eosinophilic granuloma was a variant of the processes of Hand-Schüller-Christian disease and Letterer-Siwe disease. This finding relegated these three processes—the "three sisters"—into a common category. Recent work has shown that the etiological factor may be *Paracolobactrum arizonae*, and many patients have been cured by appropriate antibiotics. Dr. Rountree and Captain Rockwood referred to visceral enlargements and stressed the overlapping characteristics of eosinophilic granuloma and Hand-Schüller-Christian and Letterer-Siwe disease.

The authors presented criteria for the differential diagnosis on the basis of history, physical and roentgenographic appearance, and pathology. They divided the pathological changes of this specific lesion into (1) the early or destructive stage, (2) the intermediate stage, with decrease in the number of eosinophils, and (3) the late or healing stage.

Sixty-three cases from the literature were analyzed, and Captain Rockwood and Dr. Rountree added six new cases of their own. The focus of the lesion was single in 78 per cent of the cases and was multiple in 22 per cent. The bones most commonly involved were the ribs, skull, vertebrae, and femur.

The authors recommended biopsy to establish the diagnosis of the lesion and treatment by curettage or radiation, or both, with support of the spine in certain cases. They discussed the relationship of eosinophilic granuloma to vertebra plana, and showed roentgenograms revealing the early lytic lesion, followed by collapse of the vertebra to the so-called silver-dollar vertebra, with preservation of the intervertebral-disc space above and below the involved vertebra so that there are no neurological signs or symptoms.

Sarcoma in Paget's Disease of Bone. Mr. Hugh C. Barry [11] said that Paget's disease of bone is more common in Australia than in many other countries, including the United States. It is extremely rare in China and other Asian countries. In the Australian community the disease is widespread in people over the age of forty years. Those persons who are admitted to the hospital are usually suffering from fractures of the femur that typically occur in the subtrochanteric region. In most cases, however, the disease remains unrecognized during the person's lifetime. The incidence of sarcoma developing in bone affected by Paget's disease is very low in Australia—perhaps less than 1 per cent. Twenty-nine cases of sarcoma of this type were treated in the four teaching hospitals in Sydney during a ten-year period.

In this series it was noted that nine sarcomata developed in the shoulder girdle, although this part of the skeleton is not usually affected by Paget's disease until the disease is extensive. Tumors developed in the skull in seven patients. It was noted that multiple primary malignant lesions appeared to be fairly common.

There was no evidence that the malignant change was precipitated by trauma, although a

8. 435 North Roxbury Drive, Beverly Hills, California.
9. 525 N.W. 11th Street, Oklahoma City 3, Oklahoma.
10. 1817 Westminster Place, Oklahoma City, Oklahoma.
11. 135, Macquarie Street, Sydney, N.S. Wales, Australia.

pathological fracture was sometimes the first sign of the disease. No case of sarcoma was noted after operative procedures, such as the insertion of a Smith-Petersen pin, intramedullary nail, or bone graft, or osteotomy or arthrodesis of the knee.

The sarcomata developing in Paget's disease varied widely as to pathology, although most were classified as osteogenic sarcomata. One patient with chondrosarcoma of the shoulder and one with a giant-cell tumor of the tibia were described. The latter patient is alive and well nearly three years after amputation. Another patient with a tumor of the popliteal space that was undiagnosed responded to deep x-ray therapy and lived for nearly eight years. A patient with sclerosing osteogenic sarcoma of the upper end of the left humerus survived for three years and eight months. Apart from these few exceptions, however, the patients showed little response to any form of treatment and most died within six months.

The Pathology of the Pseudofracture and Its Correlation with Clinical Disease. DR. IRA H. RAPP [12] said that biochemical and histological data from two clinical cases followed over a period of ten years indicated that Milkman's syndrome is a form of osteomalacia and that clinical progression and regression can be measured by biochemical surveys. The pseudofracture represents the healing of a stress area by osteoid tissue wherein conditions are not favorable for mineralization. When conditions are made suitable for calcification by appropriate treatment, pseudofractures will heal, and laboratory and roentgenographic evidence of osteomalacia will disappear. Renal dysfunction, particularly of the renal tubular absorptive mechanism, is emphasized as a cause of osteomalacia. An indiscriminate selection of a site for bone biopsy will not reveal histological evidence of osteomalacia unless a local stimulus for osteoblastic activity exists. In biopsy specimens, osteoid borders of trabeculae can only be made visible by careful decalcification and staining.

Diagnostic Problems Raised by Myositis Ossificans and Certain Other Lesions. DR. HENRY L. JAFFE [13] said that diagnostic problems may be raised by solitary radiopaque lesions presenting in the soft parts overlying or abutting a bone or joint. He excluded heterotopic ossification appearing as a sequel to paraplegia, poliomyelitis, encephalitis, meningitis, extensive burns, or progressive myositis ossificans.

Such solitary lesions may be foci of myositis ossificans or may be any of a large number of other conditions, such as: foci of necrosis with calcification; hemangiomata containing phleboliths; benign or malignant cartilage tumors undergoing focal calcification or ossification, or both; benign or malignant fibrous tumors containing foci of metaplastic bone; synoviomata containing calcospherites; osteogenic sarcomata; or lipomata whose capsules are somewhat ossified. He pointed out that these various other lesions do not often raise problems of differential diagnosis from myositis ossificans clinically or roentgenographically, and need never raise them anatomically.

Dr. Jaffe said that classical myositis ossificans is a focus of ossification in a soft part overlying, and perhaps even adjacent to, a bone or joint but not intimately adherent to the bone. When one is actually dealing with myositis ossificans, the clinical aspects of the case usually dovetail with the roentgenographic and pathological findings.

A history of severe local trauma may be elicited, but there is often no history of such trauma. If the part is easily accessible to palpation, a local painful tissue-swelling or mass may be felt within a few days after the trauma, but in the absence of a history of direct trauma, the soft-tissue mass only becomes palpable some days after the onset of local pain.

Within about a week after the onset of the clinical difficulties, there still may not be a radiopaque shadow in the soft parts. However, within another week or so, such a shadow does appear. It progressively enlarges and becomes more and more sharply delineated and usually reaches its maximum size in about two months. It then shows less radiopacity in its central portion than at its periphery.

The pathological findings reveal the clear-cut peripheral delimitation of a focus of myositis ossificans. The osseous tissue at the periphery is usually the most mature part of the lesion and the part in which its benign character stands out. Any muscle about the bone shell of the lesional area is compressed, but shows no invasion by tracts of connective tissue extending into it from the ossifying focus, such as commonly occurs adjacent to a soft-tissue osteogenic sarcoma.

In the interior of a focus of myositis ossificans that has not reached full maturity, one may see large fields containing proliferating spindle cells, osteoid, osseous tissue, and even cartilage, and also residua of degenerating muscle fibers. However, any ominous-looking cytological fields encountered in the interior of a myositis ossificans focus can safely be discounted because the lesion

12. 1500 Elizabeth Avenue, Charlotte 4, North Carolina.
13. Hospital for Joint Diseases, 1919 Madison Avenue, New York 35, N. Y.

has evolved rapidly but is well circumscribed, and presents no ominous cytological features peripherally.

Nevertheless, the distinctions between myositis ossificans and osteogenic sarcoma deserve some emphasis. In contrast to a focus of myositis ossificans, a soft-tissue osteogenic sarcoma is not infrequently a rather large lesion at the time when the patient comes under surgical care. The areas of radiopacity vary in degree and extent from lesion to lesion and, whether slight or considerable, they are more prominent in the interior of the lesion than at its circumference, often streaking out from the interior toward the periphery. The lesion is not sharply outlined by a zone of radiopacity, as a focus of myositis ossificans soon comes to be. Correspondingly, the periphery of a soft-tissue osteogenic sarcoma histologically fails to show a delimiting shell of maturing osseous tissue.

Some cases of myositis ossificans were not only in contact with bone but also altered the cortex. If the cortex of the bone becomes altered, there can be little doubt that the alteration is due to direct or indirect involvement of the regional periosteum.

The periosteum may deposit new bone because, in the process of organization, the focus of myositis ossificans may become intimately attached to the periosteum. This is what is meant by indirect implication of the regional periosteum. On the other hand, particularly if there has been a severe injury, the local periosteum may be directly implicated, having been damaged along with the overlying muscle so that the focus of ossification represents the summation of new-bone formation by the periosteum and heterotopic bone formation within the damaged muscle.

Tissue sections should be prepared from blocks of tissue that clearly include the periphery of the lesional area. The interior of the lesion may show the fields of proliferating spindle cells with osteoid and cartilage, which might raise the question of a malignant condition. However, in regard to these lesions, too, such ominous-looking cytological fields can safely be discounted if the periphery of the lesional area is outlined by mature and maturing trabeculae.

Differential diagnosis of a focus of myositis ossificans in a parosteal location must include the so-called juxtacortical osteogenic sarcoma, ordinarily a lesion of low malignancy that still is not infrequently misinterpreted roentgenographically and even anatomically as a peculiar expression of myositis ossificans. The juxtacortical osteogenic sarcoma fails to show the sharp peripheral delineation by a shell of osseous tissue and is a much more highly ossified lesion, whose osseous tissue is set in a cellular fibrosarcomatous stroma. Furthermore, the juxtacortical osteogenic sarcoma has a strong tendency toward recurrence, and in the recurrences its stromal tissue tend to show an increasingly clear-cut sarcomatous histological pattern.

Once in a while, a fully malignant soft-tissue osteogenic sarcoma develops parosteally, and such a lesion, too, may raise the question of differential diagnosis from a focus of myositis ossificans, at least on a roentgenographic basis.

Prognosis of Fractures of the Acetabulum. Dr. Carter R. Rowe [14] and Dr. J. Drennan Lowell [15] reported on the prognosis of fractures of the acetabulum from their observations of a series of ninety-three consecutive patients with such injuries admitted to the Massachusetts General Hospital. Fifty-one patients had been followed for from one to five years, and forty-two from six to twenty-seven years. They classified these patients into four groups, based on the original epiphyseal divisions of the acetabulum, because they found this method of separation clear cut and useful in the prediction of end results. Group I, patients with undisplaced linear fractures, had excellent results without late degenerative arthritis. Patients in Group II, with posterior acetabular fractures, had excellent-to-good hips in 65 per cent, and 35 per cent had fair-to-poor hips, depending on the degree of joint stability and amount of injury to the femoral head. Patients in Group III, with inner-wall fractures, were found to have excellent-to-good hips in 90 per cent, with only 10 per cent with fair, and none with poor, hips. Ninety-three per cent of this group was treated by closed conservative methods. The authors therefore feel that reduction of the inner wall is unnecessary so long as the femoral head remains under an intact superior articular dome. Patients in Group IV, with superior-dome and bursting fractures, had excellent-to-good joint function in 58 per cent and in 42 per cent the results were fair to poor. The outcome in this group was more difficult to predict, but seemed to depend on the restoration of a reduced or congruous articular surface for the superior dome and a proper relationship of the femoral head with respect to the superior dome. The authors thought that open reduction for this group should be reserved for those fractures in which the superior dome is appreciably displaced and not sufficiently improved by closed treatment. The fragments should also be of such size that they can be manipulated and fixed to restore congruity. Otherwise, initial conservative treatment appears to be preferable, with reconstructive procedures performed at a later date if needed.

14. 275 Charles Street, Boston 14, Massachusetts.
15. 275 Charles Street, Boston 14, Massachusetts.

In their survey of this entire series, the authors found that when, at the one-year follow-up examination, the hip was graded *excellent* or *good*, the outcome years later was usually excellent. They could also identify, by the end of the first year, 91 per cent of the patients who would eventually have avascular necrosis and arthritis of the femoral head.

Three cup arthroplasties were performed shortly after initial injury, and five cup arthroplasties were performed for late changes in this series. Two arthroplasties were revised because of myositis ossificans, and, in one patient with bilateral cup arthroplasties, Moore prostheses were inserted because of avascular necrosis. Two patients had spontaneous fusion of the hip, and two had surgical fusion. Traumatic arthritis and avascular necrosis of the femoral head occurred in 26 per cent of the cases. Traumatic arthritis was more common in the bursting fractures, and avascular necrosis more frequent in the posterior acetabular fractures. Myositis ossificans developed in 15 per cent of the cases—in 5 per cent of the closed-treatment groups, and in 34 per cent of the groups who had treatment by open surgery. Damage to the head of the femur was known initially in thirteen patients, eight of whom had posterior acetabular fractures. All but three of them eventually had poor hips. Other complications of these fractures included laceration of the femoral vein, thrombophlebitis, a ruptured urethra, a lacerated bladder, and a fat embolism.

Open Reduction and Traction in the Treatment of Fracture-Dislocation of the Thoracic and Thoracolumbar Vertebrae with Spinal-Cord Injury. Dr. J. Gordon Petrie [16] described the methods that have been developed with Dr. Cone and Dr. Elridge over the past twenty years, at the Montreal Neurological Institute and the Royal Victoria Hospital, for treatment of fracture-dislocations of the thoracic spine associated with spinal-cord injury. He stressed the importance of the earliest possible decompression of the injured cord, open reduction, and fixation of the fracture—that is, as soon as the patient is a safe operative risk. It was found that spinal-fluid pressure and flow measurements were of little assistance in the diagnosis of these acute injuries, and roentgenograms often failed to reveal as many fractures as were found at operation. To obtain reduction, Dr. Petrie and his associates often apply traction of from ten to 100 pounds for brief periods (up to ten minutes), using a special operating table, attached traction frames, conductive rubber mattresses with rollers, a special canvas shoulder sling, ankle boots, and pulleys, and, if the lesion is above the fourth thoracic vertebra, Cone-Barton skull tongs. They recommend removal of fragments of fractured laminae, and removal of interspinous ligaments and ligamentum flavum, or performance of a partial laminectomy for decompression of the cord if there is no laminal fracture. If an intervertebral disc is ruptured, they remove it thoroughly and curette the cartilaginous plates to encourage interbody fusion. They do not ordinarily open the dura, believing that it does not aid recovery. The procedures are usually done under local or endotracheal anesthesia after careful positioning. After thorough decompression, traction is usually applied to obtain reduction of dislocated facets in conjunction with local gentle levering or by removing small edges of the facets. After realignment, the spine is moderately extended and, if the spinous processes are intact, they are wired together. If more than two vertebrae must be fused, or if the spinous processes are removed, parallel bone grafts are wired to the intact segments and below.

Postoperative care is much simplified because internal fixation usually avoids the need for a cast. A turning frame is rotated hourly, and tidal drainage is begun at once to develop bladder control; early physical therapy and rehabilitation are also given.

On the basis of their experience with eighty cases, they thought that continued compression of the spinal cord after injury could cause progressive destruction of nerve tissue. Of their fifty-seven patients with complete paraplegia, eighteen had partial motor and sensory return. All twenty patients with partial paraplegia showed improvement in motor power and sensation. There were three deaths—one from shock, one from multiple injuries other than the spinal-cord lesion, and a third from pulmonary embolism.

Diaphyseal Fractures of the Major Long Bones. Dr. Floyd Jergesen [17] reported the results of a clinical study of 143 diaphyseal fractures in 126 consecutive patients treated by open reduction and fixation with plates and screws. All patients were treated by the author during a ten-year period. The study was designed to determine the influence of local rest or immobilization of fracture fragments on the rate and mechanism of healing of diaphyseal fractures. Two stages of bone repair were recognized and defined—restoration of bone continuity and bone healing. Restoration of bone continuity was determined by the fulfillment of the following criteria:

1. Use of the extremity for tasks common to a person of sedentary occupation. In case of the lower extremity, unsupported ambulation;

2. Absence of pain at the fracture site during use of the extremity or at stress examination;

16. 1374 Sherbrooke Street, West, Montreal 25, Quebec, Canada.
17. 384 Post Street, San Francisco 8, California.

3. Lack of radiologic evidence of displacement of fragments or fixation apparatus following operation;

4. Progressive obliteration of fracture clefts by mineralized callus, demonstrated by sequential roentgenographic examinations.

Completion of bone healing was judged by roentgenographic examination: complete obliteration of all fracture clefts; restoration of the medullary cavity; and reconstruction of cortices by remodeling and reorientation of the lamellar pattern. For purposes of comparison, a standard time for bone healing was determined from observations of recent uncomplicated fractures without comminution that had been treated with open reduction and bone plating. This standard was one year for fractures of the humerus, radius, ulna, femur, and tibia.

The rate of bone healing was found to be influenced unfavorably by local infection, reduced vascularity or non-viability of bone fragments, excessive stress on the fracture site before restoration of bone continuity, whether or not failure of the fixation device occurred, and pre-existing changes in bone, such as loss of substance, sclerosis, exuberant external callus, and obliteration of the marrow cavity.

Fourteen undisplaced fractures with delay of healing of one to nineteen years' duration were treated simply by plating without disturbing the soft tissue between the bone ends and with minimum periosteal elevation. Restoration of bone continuity occurred among twelve bones where excessive stress was avoided but complete bone healing was delayed and required one to more than five years. Two patients persisted in weight-bearing on fractured tibiae before restoration of bone continuity and healing did not occur. In the bones that healed, it appeared that dormant or arrested healing had been activated by operation, by immobilization afforded by internal fixation, or by the combined effect of both factors. Restoration of bone continuity occurred, but bone healing was markedly delayed after plating with extensive apparatus in certain fractures in the forearm with prolonged delay of healing, associated with bone atrophy. In some fractures after healing had occurred the plates and screws appeared to act mechanically as an integral part of the bone so far as structural integrity of the system was concerned. Manifestations of this were failure of the cortex to hypertrophy after obliteration of the fracture cleft and restoration of the medullary canal; refracture as the result of minimal stress after removal of the plates and screws (five cases); and breakage of screws after restoration of continuity or even after bone healing. All of the observations suggested that the fixation apparatus had continued to accept some of the applied stress.

Restoration of bone continuity was considered to be an arbitrary and poorly defined end-point of a broad intermediate phase of fracture repair. Its identification was of greatest value for practical purposes of initiating rapidly increasing graded increments of stress of the healing fracture.

Complete Dislocation and Subluxation of the Acromioclavicular Joint: End Result in Seventy Cases. Dr. Marco A. Lazcano [18], Dr. Sanford H. Anzel [19], and Dr. Patrick J. Kelly [20] evaluated the end results of forty-three complete dislocations and thirty-seven subluxations of the acromioclavicular joint seen at the Mayo Clinic from 1945 through 1957. Significant follow-up data were available on seventy of the patients. They arrived at the following recommendations and conclusions:

1. For the patient with complete dislocation of the acromioclavicular joint that is more than fourteen to twenty-one days old, the recommended treatment, if he has symptoms, is excision of the distal portion of the clavicle;

2. The results from fixation with a coracoclavicular screw in fresh or recent dislocations were excellent in four of seven patients. The method had the disadvantage of requiring meticulous technique and the later removal of the screw;

3. Fixation of the completely dislocated acromioclavicular joint by wires, screws, pins, or fascia placed across the acromioclavicular joint is not recommended, since only six of the twelve patients (excluding two patients who were lost to follow-up) had excellent results;

4. Ligamentopexy, with or without fixation with a coracoclavicular screw, when excising the outer end of the clavicle seems superfluous;

5. In acute injuries, closed reduction of the dislocation and fixation with a suspension type of cast gave excellent results in four cases, and may be recommended; however, if this is not successful, the outer end of the clavicle could be excised;

6. In subluxations, the preferred treatment is immobilization for four weeks; the results seem related to the age of the patient rather than to the mode of treatment.

18. Mayo Clinic, Rochester, Minnesota.
19. Shaw Air Force Base, South Carolina.
20. Mayo Clinic, Rochester, Minnesota.

Injuries of the Medial Ligaments of the Ankle—Result Study. Dr. O. Sherwin Staples [21] (J. Bone and Joint Surg., **42-A**: 1287–1307, Dec. 1960).

Presidential Address: The Closed Fracture of the Long Bones. Dr. John R. Moore [22] (J. Bone and Joint Surg., **42-A**: 869–874, July 1960).

Anterior Chamber Angle of the Eye in Patients with Idiopathic Scoliosis. Dr. Ignacio V. Ponseti [23], Dr. Gunter K. von Noorden [24], and Dr. Hermann M. Burian [25] said that incomplete separation between the iris and trabeculum of the anterior chamber angle is noted in patients with Marfan's syndrome. With the gonioscopic prism the pectinate ligaments formed by fine strands of tissue can be observed bridging the chamber angle from the iris to the trabecular tissue. Peripheral mounds or knotty elevations of iris tissue near the root were regarded as a lesser anomaly than the pectinate ligaments. Similar abnormalities have been demonstrated in the chamber angle in thirty-two patients with typical idiopathic scoliosis. A close correlation was found between the severity of the lesions in the chamber angle and the severity of the idiopathic scoliosis. These anomalies are absent or minimal in children with paralytic scoliosis. However, chamber-angle abnormalities were observed in the eyes of patients with slipped upper femoral epiphysis and Legg-Perthes disease.

Gonioscopic examinations of forty-two medical students revealed the presence of some degree of abnormality of the chamber angle in seventeen. A slight abnormality of skeletal development was observed in all of these seventeen cases. Students with a normal chamber angle had a well developed skeleton.

Karl Meyer reported marked decrease in chondroitin sulphate in relation with keratosulphate in the costal cartilage of patients with Marfan's syndrome.

It is suggested that Marfan's syndrome, idiopathic scoliosis, and slipped upper femoral epiphysis are expressions of an inborn error of mucopolysaccharide metabolism.

The Problem of the Primary Curve. Dr. John R. Cobb [26] (J. Bone and Joint Surg., **42-A**: 1413–1425, Dec. 1960).

Surgical Instrumentation for Management of Scoliosis. Dr. Paul R. Harrington [27] said that the instrumentation, as developed, is applicable to the axial skeleton through the conventional posterior surgical approach and accomplishes correction of multiple curves by combinations of compression and distraction forces applied to the posterior elements of the vertebrae. Experience derived from 103 cases in which instrumentation was used with and without fusion is the basis for this paper.

The distraction force is always applied to the concave side of the axial skeletal curve. The compression force is always applied to the convex side of the major curve. The forces of compression and distraction are applied through a rod that has a ratchet function for adjustment in length with the bone-purchasing hook. By surgical intervention at one-year or two-year intervals, progressive correction can be accomplished to adjust for growth changes. When the patient is ten years of age or older, spine fusion of the major curve is recommended with the initial corrective surgery.

A simple formula was developed to determine criteria of instrument application: X equals the number of vertebrae in the scoliotic curve; the degrees of curve divided by X equals a factor. When the factor is 5 or greater, instrumentation is indicated*.

Instrumentation of the axial skeleton for correction and stabilization of scoliosis as recommended here is without serious complications and affords a means to assist the orthopaedic surgeon in attacking a devastating deformity.

* A ten-year-old child whose scoliotic curve by Cobb measurement is 35 degrees and involves eight vertebrae produces factor 4.4 (35/8 equals 4.4).

Birth Injuries of the Brachial Plexus. Dr. Jack Wickstrom [28] reviewed eighty-seven patients with birth injuries of the brachial plexus. Fifty-four of them presented evidence of involvement of the fifth and sixth cervical-nerve roots. Eleven had the principal involvement of the eighth

21. 2 Maynard Street, Hanover, New Hampshire.
22. 3401 North Broad Street, Philadelphia 40, Pennsylvania.
23. State University of Iowa, Iowa Citv, Iowa.
24. State University of Iowa, Iowa City, Iowa.
25. State University of Iowa, Iowa City, Iowa.
26. 333 East 43rd Street, New York 17, N. Y.
27. 4520 Rossmoyne Boulevard, Houston 6, Texas.
28. 1430 Tulane Avenue, New Orleans 12, Louisiana.

cervical and the first thoracic-nerve roots, and twenty-two had evidence of some involvement of the entire plexus. Clinical evaluation was the sole criterion for the extent of damage. Surgical exploration of the plexus was not performed in any of the cases presented. The initial treatment consisted principally in protecting the extremity by pinning the sleeve to the cap in the immediate postnatal period, followed by the use of a Fairbank splint for approximately two to four months. This was removed for physical therapy. Excessive splinting was noted to produce an abduction contracture of the shoulder. Therefore, modifications in the position and time of splinting were made. This splint kept the arm at 70 degrees of abduction and complete external rotation, but with 45 degrees of flexion and the elbow flexed to 120 degrees. Posterior subluxation of the shoulder joint was not noted.

The results of this non-operative regimen were excellent in ten patients and satisfactory in thirty-two; thirty-three patients had poor return of function of the extremity.

The prognosis was difficult to estimate at the beginning of therapy; it was found that those patients who demonstrated a loss of thoracoscapular muscle, Horner's syndrome, or marked sensory loss had the poorest return of function. The residual deformities of limitation of external rotation of the shoulder and weakness of abduction were discussed. Residual deformities about the elbow were due to muscle weakness, and disruption of the radial humeral joint with dislocation of the radial head commonly occurred.

The various methods of reconstructive surgery were presented, and Sever's procedure for subscapular release was not so satisfactory as L'Episcopo's restoration of muscle balance by transplant of the teres major and latissimus dorsi into the external rotators. Osteotomy of the humerus above the deltoid insertion gave satisfactory functional improvement in abduction.

Surgery at the elbow to correct impaired flexion was generally satisfactory, using the Steindler flexorplasty, transplant of the triceps, and transplant of the biceps into the triceps. The results after varied reconstructive procedures to improve hand function were, on the whole, less satisfactory than for similar deformities occurring in poliomyelitis or other peripheral-nerve lesions. A variety of wrist arthrodeses and transfers were presented for each individual case, but the procedures for the finer movements of the hand and wrist were, on the whole, less satisfactory than for the gross motions of the shoulder and elbow.

Dr. Wickstrom also evaluated dissections of the brachial plexus in twenty-one stillborn infants and noted prefixation in 20 per cent as compared to 6 per cent postfixation. He did not believe that this was of any clinical significance, however. The experimental studies on the tensile strength of the brachial plexus revealed that less weight was necessary to disrupt the lower plexus than the upper plexus, and that the soft-tissue structures surrounding the plexus contributed to the resistance to stretch of the tissue. Most of the disruptions were noted at the foramen or within the groove of the transverse processes. There was clinical evidence of cord disruption, suggesting correlation with the clinically noted upper-motor-neuron lesions often seen with brachial-plexus injuries.

Hip Pathology in Cerebral Palsy. Dr. Lenox Baker [29], Dr. Richard Dodelin [30], and Dr. Frank H. Bassett [31] reported their observations on 129 patients with cerebral palsy. There was a valgus deformity of the neck of the femur in 107 patients, with varying amounts of anteversion. They found 160 hips with a high acetabular angle (in 129 patients), subluxation of forty-three hips (in thirty patients), thirty-one dislocations (in twenty-four patients), and thirty-one normal-appearing hips (in twenty-six patients). Dr. Baker discussed a number of possible explanations for the cause of the valgus deformities of the hip and their sequelae: (1) loss of hip abductor power may not stimulate the proper growth of the trochanteric epiphysis, (2) loss of normal weight-bearing may allow excess valgus, and (3) spasticity and overpull of the gracilis and medial hamstring muscles may weaken the abductors, which, secondarily, decrease stimulation of the trochanteric epiphysis. Their principal surgical efforts have been directed toward elimination of this latter condition. The importance of making careful routine periodic physical and, particularly, roentgenographic evaluation of the hips of cerebral-palsy patients in order to obtain early diagnosis and prevent the advancement of these disorders was emphasized. A modification of the gracilis test, termed the gracilis-hamstring test, which determines the presence and severity of spasticity and contracture of these muscles was described as follows: (1) both hips are firmly held in 90 degrees of flexion with as much abduction as can be obtained at this position; (2) while maintaining the opposite hip in this 90-degree flexion position, the extremity to be examined is placed in full flexion with as much abduction as possible; (3) the knee on the examined side is then extended as far as possible until adduction of the hip on the same side occurs; and (4) if the gracilis or medial hamstrings are responsible for this adduction, they will stand out as taut bands.

29. Duke University Hospital, Durham, North Carolina.
30. Duke University Medical Center, Durham, North Carolina.
31. Duke University Medical Center, Durham, North Carolina.

U.S. Army Photograph

MAJOR GENERAL NORMAN THOMAS KIRK

1888–1960

Norman Kirk died in Walter Reed General Hospital on August 13, 1960, of renal failure, a week or so after an operative repair of an aneurysm of the abdominal aorta. After services at the Hospital, he was buried with full military honors in Arlington National Cemetery on August 16, 1960.

General Kirk's entire active professional life was spent in the Medical Corps of the United States Army. He served in every rank from first lieutenant on up until he emerged as a major general, the Surgeon General of the United States Army. He received the degree of Doctor of Medicine from the University of Maryland in 1910, served as resident physician at the University Hospital, Baltimore, and then as clinical assistant at the United States Soldiers' Home Hospital in Washington, D. C. He was commissioned a first lieutenant in the Medical Reserve Corps in 1912 and appointed to the regular army as a first lieutenant in the Medical Corps in 1913. His first tours of duty carried him to Texas and the Panama Canal Zone. In 1916 he served at the Base Hospital at Brownsville, Texas, at the time of the United States intervention in Mexico.

During World War I, he was assigned to Camp Greenleaf, Georgia, from September 1917 until January 1919, when he was ordered to Walter Reed General Hospital in Washington, D. C., where, as a temporary lieutenant colonel, Kirk was credited with treating at least one-third of the major amputations incurred by the troops serving in World War I. From this and subsequent ex-

perience he derived material for a volume on amputations and operative technique, which was published in 1924.

Between World War I and World War II, General Kirk had brief tours of duty at Johns Hopkins University Hospital and at Massachusetts General Hospital. From 1925 to 1928 he served at the Station Hospital, Fort Sam Houston, Texas, as the Chief of the Orthopaedic Section and as the Assistant Chief of the Surgical Service. After this he served at Sternberg General Hospital, Manila, Philippine Islands, from 1928 to 1930, and again in 1935; at the Station Hospital, Fort Mills, Corregidor, Philippine Islands, from 1934 to 1935; and at Letterman General Hospital, San Francisco, from 1936 to 1941. He had three tours of duty at Walter Reed General Hospital (1919 to 1925, 1930 to 1934, and 1941 to 1942). During the second of these tours he passed the advanced course at the Medical Field Service School at Carlisle Barracks, Pennsylvania. His early assignments were as the Chief of the Orthopaedic Section, and later he was the Chief of the Surgical Service.

General Kirk's last hospital service was as the Commanding Officer of Percy Jones General Hospital, Battle Creek, Michigan. By this time he had been promoted to brigadier general (temporary). Under his supervision the Battle Creek Sanitorium was converted into an army general hospital, expanding from 1,750 beds to a hospital center of 12,000 beds, for the care of amputations, neurosurgery, deep radium therapy, and neurology. He also was concerned with the training of a large professional staff recruited from civilian life.

His final tour of duty was as the Surgeon General of the United States Army from June 1, 1943 to June 1947, when he retired from active service. This period of service during World War II covered the major military efforts of the United States during that war.

The Medical Corps of the United States Army during World War II expanded from 1,200 in the regular corps to 47,000 physicians, 15,000 dentists, 57,000 nurses, 18,700 administrators, 2,900 dietitians and physical therapists, 4,000 veterinarians, pharmacy, and Sanitary Corps officers, and over a half a million enlisted personnel, serving in hospitals as medical-aid men, litter-bearers, ambulance drivers, and technicians. This enormously expanded personnel manned 1,200 separate hospitals with a 750,000 bed capacity in all theaters of operation and the Zone of the Interior, furnished consultants in all branches of medicine in most of the areas where our troops were deployed, and took care of 15,000,000 hospital admissions.

Under General Kirk's guidance, the Army's ground and air forces during World War II achieved a record of recovery from wounds and freedom from disease never before accomplished in military medical history. The death rate from disease was 60 per 10,000, compared with 165 per 10,000 in World War I. There was no case of yellow fever, and only five deaths from tetanus were recorded. Our troops who fought where cholera, typhus, and plague were endemic almost entirely escaped these diseases. The incidence of malaria and dysentery—diseases that have decimated our armies in previous wars—were held under control. Meningitis and pneumonia, with a mortality of 38 and 24 per cent, respectively, in World War I, during World War II showed mortality rates of 4 and 0.6 per cent.

These figures, for our troops, reflect the progress of civilian medicine over the years. The achievement of the Army Medical Corps under General Kirk was that the finest and most current medical and preventive medical care was afforded promptly to our troops in all army hospitals and theaters of operation.

During this war some methods of surgical treatment were employed on an enormous scale that had no counterpart in civilian surgery between the two world wars. After adequate débridement, delayed primary closure of wounds converted open fractures into simple fractures with enhanced healing. Amputation of an extremity was performed only for irretrievable damage, complete loss of blood supply, or clostridial myositis. The fact that there were only approximately 17,000 major amputees among all the United States service personnel with a total mobilization of about 14,000,000 speaks eloquently of the salvage effected on gunshot wounds of the extremities. Thoracic and abdominal surgery were made available near the scene of action. Neurosurgical procedures were performed as indicated in the theaters of operation. Treatment of shock and hemorrhage with whole blood made available in large quantities saved countless lives. Penicillin and sulfonamides played some part in minimizing infection, but prompt and definitive surgery was mainly responsible for the fact that 96 out of every 100 wounded, who lived to reach a hospital, survived, and that 376,000 of 598,000 wounded were returned to duty in the various theaters of operation overseas, and an additional 35,000 returned to duty in the United States.

This record is a measure of the work of large numbers of well trained surgeons who were intelligently directed and strategically placed under the guidance of General Kirk.

Norman Kirk was a surgeon of great ability, whose first interest was in orthopaedic surgery. He later turned to, and became proficient in, general surgery. His proficiency in orthopaedic surgery was recognized by The American Orthopaedic Association, which elected him to membership in 1929 when he was a major and was stationed at Sternberg General Hospital in Manila. He was

elected to the American Surgical Association somewhat later but still some time before he became the Surgeon General.

To some of his colleagues who knew him but slightly as the Surgeon General, he may have seemed brusque, tough, or even hard-boiled. The writer has turned to two of Kirk's more intimate friends who served with him. Major General Raymond Bliss, Medical Corps, United States Army, Retired, who was a life-long friend and succeeded Kirk as the Surgeon General, wrote as follows:

"A great deal of ruthlessness, or intolerance, manifested itself in Norman due to long association with Colonel Keller, a perfectionist, a fine surgeon, completely devoted to his patients, but tough on the ignorant or careless doctor. In his almost puritanical zeal, Kirk could not condone professional ignorance nor any breach of duty. He was more than a strict disciplinarian, a man of complete integrity, always doing more than he expected of others. He had a long memory and was perhaps sparing in charity towards officers he considered to have been derelict or ignorant of proper professional standards. His patients loved him for his kindliness and his humanity. In all his years of hospital life, I doubt if he ever missed making rounds at night and personally checking every one of his seriously ill patients. He was an able perfectionist and thorough in every detail of his life."

Dr. Robert H. Kennedy, who was a colonel in the Army of the United States, was under General Kirk at Percy Jones General Hospital; he wrote of Norman Kirk as follows:

"There was never enough he could do for the sick or wounded soldier, or for that matter, for the rank and file of G.I.s. He would not tolerate a man giving anything short of his best professional service. Intellectual dishonesty usually meant a quick transfer. He was kind to human weaknesses and a most loyal friend in times of trouble, as many officers and men of all ranks can testify."

It is well for all of us to contemplate and take a measure of pride in these tributes to a man under whom many of us had the privilege of serving. Among the great successes in his career in the United States Army Medical Corps, there were two bitter disappointments, which he surmounted. In World War I, he was not sent overseas to serve with the Expeditionary Forces, and, in World War II, at the outbreak, he was considered too old for service with our Armed Forces overseas. As the Surgeon General he made three overseas inspection tours with the Secretary of War.

General Kirk was a diplomate in both orthopaedic and general surgery. He was a fellow of the American College of Surgeons, and also the American College of Physicians. He was a member of the Southern Surgical Association, the American Association for the Surgery of Trauma, the American Medical Association, the American Hospital Association, the National Research Council, and The American Academy of Orthopaedic Surgeons, among others.

Davidson College and the University of Maryland conferred the honorary degree of Doctor of Science, and Columbia University conferred the degree of Doctor of Laws on General Kirk.

He was awarded the Legion of Merit and the Distinguished Service Medal by the United States. He held the honorary rank of Commander, Order of the British Empire, the French Cross of the Legion of Honour, and that of Commander, Order of the Crown of Italy.

General Kirk's medical bibliography contains eighteen publications, including the book on amputations. The great majority of these contributions were on orthopaedic subjects, army medical problems, and subjects related to trauma.

His family life was a happy one. His wife, Anne Duryea Kirk, whom he married in 1915, two daughters, Anne Kirk Willard and Jane Kirk Kimbrell, and five grandchildren survive him.

Norman Kirk loved good company with his friends. All his life he was an avid fisherman and hunter. After his retirement he was able to indulge in fishing at his home at Montauk, Long Island, and at his winter home in Melbourne Beach, Florida. He spent a good deal of time and loving care on his plants, flowers, and vegetables. As General Bliss remarked: "His garden without a weed had all the plants lined up like soldiers on parade".

He approached his impending operation for abdominal aortic aneurysm last August with hope and without fear. He waited until after a visit from his grandchildren, I am told. It is fitting that we take courage and satisfaction from such a life of completely dedicated service.

M. C.

Book Reviews

ANATOMY: A REGIONAL STUDY OF HUMAN STRUCTURE. Ernest Gardner, M.D., Donald J. Gray, Ph.D., and Ronan O'Rahilly, M.Sc., M.D. Philadelphia, W. B. Saunders Co., 1960. $15.00.

The teaching of anatomy in contemporary medical schools is plagued by lack of allocated time. Anatomy is still one of the most important and fundamental subjects, not only for future surgeons, but for practitioners in all branches of medicine. One of the great French neurologists of the nineteenth century stated on the front page of a great book on muscle physiology that "without anatomy there is no physiology, no surgery, no medicine". It is true that the programs of the medical schools require the inclusion of too many subjects.

It is very hard to find an immediate solution to the problem, but whatever the solution may be, anatomy must be given enough time, and a high enough priority to make sure that it is inoculated into the future physician as the great basis on which he may build his practical work and further insight into scientific development.

It is obvious that the approach to anatomy at present differs considerably from the teaching in the nineteenth and beginning of the twentieth centuries. The detailed descriptions of purely morphological significance are presently replaced by a broad combination of functional, developmental, and comparative factors that are important for understanding of the living subject and that are a part of physiology, pathology, and treatment. This can be achieved when the novice in medicine is given the opportunity for comprehensive dissection and the proper inspiration and indoctrination in the subject of functional anatomy.

The reviewer believes that the volume written by the three contributors represents a text excellently adapted to the limitations imposed by the new medical-education programs. The authors of "Anatomy: A Regional Study of Human Structure" are known for their original contributions in the broad field of anatomical investigations. They succeeded in creating a textbook that is well balanced between the functional approach, an amazing amount of important structural detail, and enough clinical application to incite further study and interest in anatomy.

The regional descriptions facilitate the proper understanding of structures and their relations. The structures of interest to the orthopaedic surgeon are treated with such lucidity in their functional and morphological aspects that this part will be of interest not only to the beginners but may serve as a useful reference to others. The results of the most recent investigations are well incorporated in the text. A special feature of great help is the inclusion of selected valuable references, providing further information and inciting the reader to continue to investigate various problems of functional anatomy. There are many original line drawings and a number of clear roentgenograms that enhance the understanding of the text.

The book is of distinct value to the medical student and should be always available to every practicing orthopaedic surgeon.

Emanuel B. Kaplan, M.D.

ATLAS DE TECHNIQUE OPÉRATOIRE. CHIRURGIE DE LA MAIN. M. Iselin, L. Gosse, S. Boussard, and D. Benoist. Paris, Flammarion, 1958.

An atlas cannot replace a textbook, but frequently it can serve as an important and necessary supplement. An atlas on operative technique permits elaboration of certain details and the introduction of refinements not always possible or even desirable in a textbook. It is especially valuable when the technical details are based on extensive personal experience. The present volume is an excellent example of what an atlas can accomplish.

There is enough description of the illustrations to make the drawings very useful and easy to follow. The book has 329 pages and 693 line drawings that are simple and comprehensive.

The subject matter covers the essential anatomy and physiology of the hand and wrist; it also covers the methods of treatment to be followed in fresh injuries to bone and soft tissues, in secondary repair after various traumatic lesions, and in various acquired afflictions of the hand, including congenital deformities of the fingers.

There are many ingenious figures showing details in transplantation of whole digits, and there are fine, simplified illustrations of plastic repair of the skin, tendons, and bone in afflictions of the hand and forearm. Simple line diagrams often illuminate details that would otherwise require lengthy descriptions. Many original ideas of approach and technique are found throughout the pages of the book. The authors pay tribute to Sterling Bunnell and to his management of tendons and other structures, as well as to other American and Continental authorities.

The book is replete with ingenious devices and suggestions that could be of great help in the

treatment of many complicated injuries. Verdan's method of extra-osseous subcutaneous wire splinting for fixation of phalangeal fractures is illustrated, as is the use of Reverdin's needle in some cases where it is of value in hand surgery. Only a few of the valuable points mentioned in this book are mentioned in this review to illustrate the general scope of the book.

This is a delightful and valuable volume that could be used profitably by the master hand surgeon as well as by the beginner, even without knowledge of French.

Emanuel Kaplan, M.D.

METABOLIC CARE OF THE SURGICAL PATIENT. Francis D. Moore, M.D. Philadelphia and London, W. B. Saunders Co., 1959. $20.00.

The author, as Professor of Surgery of the Harvard Medical School and Surgeon-in-Chief of the Peter Bent Brigham Hospital, is eminently qualified to write this volume on the current knowledge of metabolism and its surgical applications.

This work is divided into six parts, each housing its own table of contents, systematic discussion, notes from the literature, illustrations, clinical procedure, summaries, and case records.

These parts are entitled as follows: (1) The Normal Patient, and the Metabolism of Recovery; (2) The Blood Volume: Hemorrhage, Plasma Loss, Transfusion, and Hypervolemia; (3) Body Fluid and Electrolyte: Water, Salt, and Acid; (4) Loss of Body Substance: Body Composition and Clinical Management in Surgical Starvation; (5) Visceral Disease in Surgical Patients; and (6) Fractures, Wounds, and Burns.

This book presents the metabolic problems encountered in surgery in a clear and concise style, with many illustrative figures and cases. Of the many sections in the book, those on the metabolism of the skeleton, fractures, and trauma will be of special interest to surgeons interested in trauma and orthopaedic surgery. This book is an outstanding contribution.

Crawford J. Campbell, M.D.

CHRISTOPHER'S TEXT BOOK OF SURGERY. Ed. 7. Edited by Loyal Davis, M.D. Philadelphia, W. B. Saunders Co., 1960. $17.00.

Christopher's Text Book of Surgery has undergone seven editions, beginning in 1936; the last one prior to the 1960 edition was copyrighted in 1956. Advances in some fields of surgery are taking place rapidly, so that a new edition four years after the previous one is well justified. In the preface one reads that this book "aims to aid the student of surgery of all ages to approach an understanding of surgical axioms properly". To achieve this, the book is organized according to, first, general approaches to the subject, and, second, regional considerations. There is a section of the history of surgery by A. O. Whipple, a section of qualifications of the surgeon, and a valuable chapter on surgical judgment. One hundred and ninety-nine pages are devoted to the bones and joints. There are chapters on urology, gynecology, and neurosurgery, as well. Completely new sections have been written on numerous subjects, and all the chapters have been reviewed and revised. The stature of the individual contributors is impressive, most of them being outstanding authorities on their subjects. A welcome addition is a brief biographical sketch on each of them. A reviewer cannot know enough about all the subjects to be able to criticize the individual sections, but when one reads over the articles on those subjects with which he is familiar, he is convinced that the chapters are well written and comprehensive. In the interests of brevity, some of the authors have had to be somewhat dogmatic in their statements about treatment of choice; this is understandable. The book is well illustrated with line drawings and photographs. Surgery has become such a vast subject that coverage in one volume is impossible, but Christopher's Text Book of Surgery comes close to achieving this in its 1515 pages. The sections of preoperative and postoperative care should be required reading for those concerned with management of the surgical patient. For the orthopaedic surgeon, dealing as he must with problems of care of the injured, this book is highly recommended.

Donald P. Shedd, M.D.

CINEFLUOROGRAPHY: PROCEEDINGS OF THE FIRST ANNUAL SYMPOSIUM ON CINEFLUOROGRAPHY, Sponsored by the Department of Radiology, University of Rochester School of Medicine and Dentistry, Rochester, New York, November 14 and 15, 1958. Edited by George H. S. Ramsey, M.D., Theodore A. Tristan, M.D., James S. Watson, Jr., M.D., Sydney Weinberg, and William S. Cornwell, M.A. Springfield, Illinois, Charles C. Thomas, 1959. $11.75.

This book is composed of a collection of papers presented in 1958 at the first annual symposium on cinefluorography sponsored by the Department of Radiology of the University of Rochester, Rochester, New York. These papers deal with the many technical aspects of the making of "x-ray movies" and discuss many of the problems encountered in the science as it was at that

time. The publication is mainly technical and makes no attempt to cover the subject of anatomical interpretation of the films and cannot be used for this purpose.

The field is well covered in the historical as well as the present-day aspects. The early part of the book deals with the past and present methods used to produce these movies and with the evolution of the image-intensifier tube, which makes the science much more practical and safe than in the past. Further chapters discuss the merits of the various sizes of image-intensifier tubes available, and the application of television to the method. Practical details concerning the type, storage, and development of film, as well as recording cameras and other equipment, are well covered in lectures by several authors. The book is well illustrated and has diagrams that are particularly useful.

Some of the book is highly technical, with mathematical formulas, and may not be of interest to the average orthopaedist. Other parts are easily understandable for the reader with little technical knowledge. The book is not recommended for general use or general reading, but it is recommended for the person who is actively engaged in the problems of cinefluorography. The discussion of the value of thirty-five-millimeter *versus* sixteen-millimeter film, as well as the discussion of the merits of the various sizes of image-intensifier tubes, is well done. This reviewer believes that sixteen-millimeter film and the smaller five-inch tube are perhaps the most practical for orthopaedic use.

Some interesting new advancements (as they existed in 1958) are discussed, among which are fiber optics, which transmit light along a cluster of small glass tubes and are said to be the most efficient means of transferring an image from one surface to another. Others include a discussion of solid-state amplifying screens, which intensify the image without the use of the image-intensifier tube.

In summary, the series of papers forming this book covers the various aspects of the science well. Its highly technical nature, without discussion of anatomical considerations, may make it inadvisable for any but those who are actively interested in this subject.

J. William Fielding, M.D.

AN ATLAS OF NEUROSURGICAL TECHNIQUES. James L. Poppen, M.D. Philadelphia and London. W. B. Saunders Co., 1960. $28.00.

This beautifully illustrated and printed book offers a step-by-step pictorial demonstration of neurosurgical procedures. It includes a wide variety of operations, from diagnostic procedures to major intracranial and spinal explorations. The pictures reveal the brilliant surgical technique of the author and his efforts to standardize even those procedures that are performed for the treatment of difficult and unpredictable pathological processes, such as the various vascular anomalies of the brain and the like. The basic principles are those accepted by most modern neurosurgeons, yet the descriptions contain many individual touches and practical time-saving but safe short cuts. Some of the individual variations will probably be disputed by other experts, but these are the very points that give this book its definite character.

The book consists of five parts, covering the operations on the head, neck, chest and abdomen, extremities, and spine, as well as the diagnostic procedures. Indications and contra-indications for surgery are not discussed. The text takes the form of legends for the illustrations. It is therefore short, succinct, and, occasionally, almost telegraphic in style, but clear. The illustrations are superb and very clear; their three-dimensional effect is sometimes striking. Several artists were employed who used different techniques of illustration. Strangely enough, this does not detract from the compactness of the illustrative material, and the lack of artistic monotony is quite pleasing.

Not all of the standard operations are depicted, but the selection covers a wide range. Some areas are lightly treated; entirely missing are the developmental anomalies of the spine, except for one standard operation on myelomeningocele. Procedures performed mostly by orthopaedic surgeons, such as the various types of spine fusions, are not included. The chapter on the diagnostic procedures is somewhat disappointing. It could be condensed. The photographs on encephalography and arteriography are out of character and do not measure up to the artistic standard of the rest of the book.

This book reflects the life experience of one of the masters of neurosurgery and will remain a standard of reference for many years to come. It should be available in every hospital where neurosurgical operations are performed.

Louis Bakay, M.D.

LEHRBUCH DER ORTHOPÄDIE. Prof. Dr. Med. Peter F. Matzen. Berlin, VEB Verlag Volk und Gesundheit, 1959. 2 vols. DM 113,50.

Since the completion in 1907 of the exhaustive *Handbuch der orthopädischen Chirurgie* edited

by Joachimsthal, there has been no attempt to publish a system of orthopaedics in Germany until the current monumental four-volume work by Hohman, Hachenbroch, and Lindmann. Three volumes had appeared by 1959. These are first-class reference books, and the German-reading surgeons eagerly await the fourth volume. The contributors include some of the foremost orthopaedists in Europe, among whom is the distinguished Professor Dr. Peter F. Matzen, of the University of Leipzig, who wrote on joint lesions for the first volume.

It is significant that in 1959 a textbook of orthopaedics was published in East Berlin with Professor Matzen as the editor. He selected a group of brilliant young contributors, including some from West Germany. The scientific value and practical importance of this work are truly representative of all of Germany. It is regrettable that the paper and the binding do not maintain the high standards of the text. Despite this, the illustrations are good. Most of them are originals and well suited to teaching at the medical-school level.

The incomparable German heritage of orthopaedic surgery at the turn of the century is well preserved in numerous brief historical sketches. The early Germanic contributions are concisely presented, debunked, and brought up to date.

The first volume of 597 pages presents the general subject of orthopaedic surgery with good chapters on methods of examination, techniques of non-operative treatment, instruments, and apparatus. Unusual in a German orthopaedic textbook is the excellent summary of the principles of fracture treatment, supplemented by a more detailed discussion of fractures in the second volume. There are inevitable duplications in the two volumes, but this opportunity for two contributors to discuss the same subject increases, rather than detracts from, the value of the text.

In the first volume, various bone, nerve, muscle, tendon, and fascial lesions are discussed. In the second volume, there is a more detailed study according to anatomical regions.

A general review of the famous German corrective gymnastics places them in their proper relationship to other orthopaedic modalities. It is refreshing to read in German that the Klatt creeping exercises will mobilize a scoliotic spine and lead to more rapid progression unless carefully administered and accompanied by appropriate support. Physical therapy and occupational therapy are carefully integrated with orthopaedic treatment in a manner which should be an inspiration to American readers. There is a clear statement of goals to be obtained by these ancillary services. The lifelong training of the patient to accept an order as sufficient motivation for intensive and sustained corrective exercises gives the German physical therapist an advantage.

Rehabilitation is primarily the job for the orthopaedic surgeon, and the intricacies of non-operative treatment are not neglected. The team approach is stressed and illustrated by experiences gained through two world wars. It helps in job placement to have a law specifying that 10 per cent of all employees of certain industries must be physically handicapped persons.

It would be wrong to single out individual chapters for their excellence. All phases of orthopaedics are included, but only in sufficient detail for the reference work of a student or a generalist. Throughout the volumes there is a most commendable tendency to correct the old German teaching that all deformities are caused primarily by ill-fitting shoes, poor posture in school, and other mechanical insults. Postural defects are well presented as they are related to, but not necessarily caused by, improper clothing and confining occupations.

Many of the techniques stem from World War II, but the historic importance of others is not neglected. The original Quengel method and the adaptation of its principle to various splints are examples of the many therapeutic tricks that need publicizing in our country.

This outstanding textbook of orthopaedics should be in every medical-school library. It is a priceless ready reference of German contributions toward the early development of orthopaedics, supplemented by modern advances in this specialty. The latter are well presented by an experienced editor aided by his disillusioned contributors.

Walter P. Blount, M.D.

THE ORTHOPAEDIC RESEARCH SOCIETY

The Annual Meeting of the Orthopaedic Research Society will be held at the Americana, Bal Harbour, Miami Beach, Florida, Saturday and Sunday, January 7 and 8, 1960. The following program has been planned:

Saturday, January 7

Microangiography in Fracture Healing. Frederic W. Rhinelander and R. A. Baragry, Cleveland, Ohio (discussed by James B. Wray, Syracuse, New York);

An Experimental Study of Joint Stiffness. Wayne H. Akeson, Chapel Hill, North Carolina (discussed by Robert S. Siffert, New York, N. Y.);

The Role of the Parosteal Tissue in the Post-traumatic Osteogenesis of Long Bones. Leo Jarry and Hans Uhthoff, Montreal, Canada (discussed by C. Andrew L. Bassett, New York, N. Y.);

Circulation of the Head of the Femur Following Fracture (in Dogs). Charles J. Frankel and Paul Derian, Charlottesville, Virginia (discussed by Irving Stein, Philadelphia, Pennsylvania);

The Production of Mandibular Fractures. A Study Using the Stresscoat Technique and High-Speed Cinematography. Donald F. Huelke, Ann Arbor, Michigan (discussed by F. Gaynor Evans, Ann Arbor, Michigan);

Induction of Heterotopic Bone Formation by Use of Extracts Made from Bone Subjected to Passive Stasis. Billie D. Burdeaux, Isaac S. McReynolds, and Kairyu Hasegawa, Houston, Texas (discussed by E. Burke Evans, Houston, Texas);

A Comparative Study of Tendon-to-Tendon and Tendon-to-Bone Suture Line Strength. Jack Levine, Abel Kenin, and Morton Spinner, Brooklyn, New York (discussed by S. Benjamin Fowler, Nashville, Tennessee);

An Experimental Study of Lymphatic Pathways of Bone, Bone Marrow, and Joints. Paul Schutt and Ernest Gardner, Detroit, Michigan (discussed by David W. Anderson, Seattle, Washington);

Localized Epiphyseal Arrest Following Intramedullary Papain Injection. Monroe Schneider, Brooklyn, New York (discussed by Wayne O. Southwick, New Haven, Connecticut);

Comparison of Ca^{45} and Tetracycline Localization in Bone. William H. Harris, Jenifer Jowsey, and Robert Jackson, Boston, Massachusetts (discussed by Thomas D. Brower, Pittsburgh, Pennsylvania).

Sunday Morning, January 8

Articular Cartilage Chondrolysis after Injection of Sterile Bacterial Enzymes. Willard S. Hunter and Sherman S. Coleman, Salt Lake City, Utah (discussed by C. Roger Sullivan, Rochester, Minnesota);

Mucopolysaccharide Production of Synovial Cells during Organ Culturing. William B. Fischer and John Torgerson, Chicago, Illinois (discussed by Robert D. Ray, Chicago, Illinois);

Acid Mucopolysaccharidurla in Heritable Disorders of Skeletal Development. Andrew E. Lorincz, Gainesville, Florida (discussed by O. Donald Chrisman, Northampton, Massachusetts);

Synovial Fluid Levels of Penicillin V after Massive Oral Administration. Ben T. Bell, Peter Viek, and Samuel C. Santangelo, Bryn Mawr, Pennsylvania (discussed by Edward Cutler, Miami, Florida).

Sunday Afternoon, January 8

Combined Program of The American Academy of Orthopaedic Surgeons and the Orthopaedic Research Society.

Pathological Changes in Osteochondrodystrophy. Carl E. Anderson, Jackson T. Crane, Harold A. Harper, and Hunter Wesley, San Francisco, California (discussed by James S. Miles, Denver, Colorado);

Some Bioelectric Aspects of Regeneration. Robert O. Becker, Syracuse, New York (discussed by Crawford J. Campbell, Albany, New York);

Evaluation of the Hellstadius Experiment. Bertram M. Kummel, Morristown, New Jersey (discussed by Edgar M. Bick, New York, N. Y.);

Post Fracture Vascular Phenomena and Long Bone Overgrowth in the Immature Skeleton. James B. Wray and Harold O. Goodman, Syracuse, New York (discussed by Frederic W. Rhinelander, Cleveland, Ohio);

Research Concerning the Repair of Knee Ligaments in Dogs. Don H. O'Donoghue and Charles M. Rockwood, Oklahoma City, Oklahoma (discussed by J. Vernon Luck, Los Angeles, California);

Studies on the Pathogenesis of Collagen Tissue Changes in Alcaptonuria. Robert A. Milch, Baltimore, Maryland (discussed by Michael Bonfiglio, Iowa City, Iowa).

INDEX TO VOLUME 42-A

(American Volume)

1960

AUTHORS

A PAGE

Abe, Yasukazu; Oshima, Naozo; Hatanaka, Ryoshin [correctly, **Hanakita, Yoshiomi**]; **Amako, Tamikazu**; and **Hirohata, Ryozo.** Thirteen Cases of Alkaptonuria from One Family Tree with Special Reference to Osteo-Arthrosis Alkaptonurica..... 817

Ackerman, Lauren V. *See* **Whitesides, Thomas E., Jr.**

Aitken, Alexander P., and **Nalebuff, Edward A.** Volar Transnavicular Perilunar Dislocation of the Carpus..... 1051

Akahoshi, Yoshihiko. *See* **Ferguson, Albert B., Jr.**

Altman, Donald H., and **Smith, Richard L.** Unrecognized Trauma in Infants and Children..... 407

Amako, Tamikazu. *See* **Abe, Yasukazu.**

Ardizzone, Joseph. *See* **Gordon, Everett J.**

Axer, Anatol. Into-Talus Transposition of Tendons for Correction of Paralytic Valgus Foot after Poliomyelitis in Children..... 1119

B

Badgley, C. E. *See* **Bailey, R. W.**

Bailey, R. W., and **Badgley, C. E.** Stabilization of the Cervical Spine by Anterior Fusion... 565

Ballinger, Charles S. *See* **Dee, Paul E.**

Banks, Henry H., and **Green, William T.** Adductor Myotomy and Obturator Neurectomy for the Correction of Adduction Contracture of the Hip in Cerebral Palsy..... 111

Bartone, Noel F. *See* **Vasilas, Anthony.**

Bechtol, Charles O. *See* **Marmor, Leonard.**

Becker, Robert O. The Electrical Response of Humal Skeletal Muscle to Passive Stretch... 1091

Bell, Ellen H. *See* **Bergstrom, William H.**

Bennett, George W. *See* **Hassler, William L.**

Bergstrom, William H., and **Bell, Ellen H.** Bone Magnesium Content in Normal and Acidotic Rats..... 437

Bickel, William H. *See* **Troup, James B.**

Blodgett, William H., and **Houtz, S. J.** Clinical and Electromyographic Evaluation of Patients with Anterior Transposition of Peroneal Tendons..... 59

Blumel, Johanna. *See* **Evans, E. Burke.**

Borgia, Charles A. A Rare Subperiosteal Metastasis Eroding the Femoral Cortex Eight Years after Radical Mastectomy for Adenocarcinoma..... 1084

Bost, Frederic C. *See* **Sutherland, David H.**

Bost, Frederic C.; Schottstaedt, Edwin R.; and **Larsen, Loren J.** Plantar Dissection. An Operation to Release the Soft Tissues in Recurrent or Recalcitrant Talipes Equinovarus..... 151

Bosworth, David M.; Fielding, J. William; Liebler, William A.; Ishizuka, Tadao; Ikeuchi, Hiroshi; and **Cohen, Paul.** Hip Shelves in Children..... 1223

Boyer, Delos W. *See* **Smith, Albert G.**

Boyes, Joseph H.; Wilson, James N.; and **Smith, James W.** Flexor-Tendon Ruptures in the Forearm and Hand..... 637

Brannon, Earl W. Congenital Malformation of the Atlanto-Axial Joint with Dislocation... 1377

Broman, George E. *See* **Trotter, Mildred.**

Bugg, Everett I., Jr. *See* **Smith, Albert G.**

Butler, James K. *See* **Evans, E. Burke.**

PAGE

C

Cahen, Irvin. *See* **Winters, Jack L.**
Canales, Gregorio M. *See* **Ford, Lee T.**
Carpenter, Earnest B., and **Garrett, Roland G.** Congenital Pseudarthrosis of the Clavicle. Report of a Case with Resection and Bone-Grafting. 337
Chandler, E. F. *See* **Woolfrey, B. F.**
Child, Charles G., III. *See* **Flynn, J. Edward.**
Chrisman, O. Donald. *See* **Coelho, Rose Robyns.**
Christie, James H. *See* **Cordrey, Lee J.**
Close, J. R.; Nickel, E. D.; and **Todd, F. N.** Motor-Unit Action-Potential Counts. Their Significance in Isometric and Isotonic Contractions. 1207
Cobb, John R. The Problem of the Primary Curve. 1413
Coelho, Rose Robyns, and **Chrisman, O. Donald.** Sulphate Metabolism in Cartilage. II. S^{35}-Sulphate Uptake and Total Sulphate in Cartilage Slices. 165
Cohen, Jonathan. Simple Bone Cysts. Studies of Cyst Fluid in Six Cases with a Theory of Pathogenesis. 609
Cohen, Jonathan, and **Foultz, W. Sanford.** Failure by Corrosion of a Steinmann Pin Used for Intramedullary Fixation. 1201
Cohen, Jonathan, and **Weinfeld, Marvin S.** Experimental Excision of Muscles in the Weanling Rat. 278
Cohen, Paul. *See* **Bosworth, David M.**
Coleman, S. S., and **Hunter, W. S.** Extra-Articular Hip Arthrodesis. Salvage Procedure for Persistent Pain Following Replacement Arthroplasty: A Case Report. 1062
Coonrad, Ralph W. *See* **Smith, Albert G.**
Cordrey, Lee J.; Christie, James H.; MacIntyre, William J.; and **Palmer, Rufus.** A Study of Strontium^{85} Metabolism in the Normal and in the Castrated Mouse. 1349
Cordrey, Lee J., and **Ferrer-Torells, Miguel.** Management of Fractures of the Greater Multangular. Report of Five Cases. 1111
Costen, William. *See* **McCarroll, H. R.**
Crawford, Henry B. Conservative Treatment of Impacted Fractures of the Femoral Neck. A Report of Fifty Cases. 471

D

Dash, Udaya N., and **Handler, David.** A Case of Compression of Subclavian Vessels by a Fractured Clavicle Treated by Excision of the First Rib. 798
Dee, Paul E.; Smith, R. Glenn; Gullickson, Miles J.; and **Ballinger, Charles S.** The Orthopaedist and Apical Lung Carcinoma. 605
Donaldson, William F. *See* **Gartland, John J.**
Doyle, James R. Narrowing of the Intervertebral-Disc Space in Children. Presumably an Infectious Lesion of the Disc. 1191
Duncan, William R. Tonic Reflexes of the Foot. Their Orthopaedic Significance in Normal Children and in Children with Cerebral Palsy. 859
Dunn, Adolphus W. Coxa Plana in Monozygotic Male Twins. 178

E

Eggers, G. W. N. *See* **Evans, E. Burke.**
Enneking, W. F. *See* **Hammack, B. Leslie.**
Evans, E. Burke; Eggers, G. W. N.; Butler, James K.; and **Blumel, Johanna.** Experimental Immobilization and Remobilization of Rat Knee Joints. 737

F

Farmer, A. W., and **Laurin, C. A.** Congenital Absence of the Fibula. 1
Ferguson, Albert B., Jr. *See* **Gartland, John J.**
Ferguson, Albert B., Jr., and **Akahoshi, Yoshihiko.** Vascular Patterns in Immobilized, Denervated, or Devascularized Rabbit Limbs. 617
Ferguson, Albert B., Jr.; Laing, Patrick G.; and **Hodge, Edwin S.** The Ionization of Metal Implants in Living Tissues. 77
Ferrer-Torells, Miguel. *See* **Cordrey, Lee J.**
Ferrer-Torells, Miguel. *See* **Furey, J. George.**
Field, Paul. *See* **Salter, Robert B.**
Fielding, J. William. *See* **Bosworth, David M.**

PAGE

Fielding, J. William; Liebler, William A.; and **Tambakis, A.** The Effect of a Tibial-Tubercle Transplant in Children on the Growth of the Upper Tibial Epiphysis. 1426

Flynn, J. Edward; Wilson, John T.; Child, Charles G., III; and **Graham, James H.** Heterogenous and Autogenous-Tendon Transplants. An Experimental Study of Preserved Bovine-Tendon Transplants in Dogs and Autogenous-Tendon Transplants in Dogs. 91

Ford, Lee T. *See* **Lansche, W. Edward.**

Ford, Lee T., and **Canales, Gregorio M.** A Study of Experimental Trauma and Attempts to Stimulate Growth of the Lower Femoral Epiphysis in Rabbits—III. 439

Fortson, Charles H. *See* **Shroyer, Russell N.**

Foster, Eugene A. *See* **Nicoll, E. D. Vere.**

Foultz, W. Sanford. *See* **Cohen, Jonathan.**

Freehafer, Alvin A. Osteochondritis Dissecans Following Legg-Calvé-Perthes Disease. A Report of One Case. 777

Freiberger, Robert. *See* **Nicholas, James A.**

Frost, Harold M. *In Vivo* Osteocyte Death. 138

Frost, Harold M. Micropetrosis. 144

Frost, Harold M. A New Bone Affection: Feathering. 447

Furey, J. George; Ferrer-Torells, Miguel; and **Reagan, James W.** Fibrosarcoma Arising at the Site of Bone Infarcts. A Report of Two Cases. 802

G

Garcia, Alexander, Jr., and **Grantham, S. Ashby.** Hematogenous Pyogenic Vertebral Osteomyelitis. 429

Garrett, Roland G. *See* **Carpenter, Earnest B.**

Gartland, John J., *Chairman;* **Ferguson, Albert B., Jr.; Donaldson, William F.; Goodman, Marvin C.; Rodriguez, Ervin E.;** and **Skovron, Michael.** Traumatic Dislocation of the Hip Joint in Children. A Report by the Scientific Research Committee of The Pennsylvania Orthopedic Society. 705

Goldenberg, Raphael R. Well Differentiated Fibrosarcoma of the Calcaneus. Report of a Case Treated by Resection. 1151

Goodman, Marvin C. *See* **Gartland, John J.**

Gordon, Everett J., and **Ardizzone, Joseph.** Clinical Experiences with the S.A.C.H. Foot Prosthesis. 226

Gottschalk, Raymond G. Radioactive Sulphur in Chondrosarcomata. The Distribution, Concentration, and Effects of S^{35} after Intravenous Administration to Patients with Chondrosarcoma. 1239

Graham, James H. *See* **Flynn, J. Edward.**

Grantham, S. Ashby. *See* **Garcia, Alexander, Jr.**

Green, William T. *See* **Banks, Henry H.**

Grieco, R. Vincent. *See* **Vasilas, Anthony.**

Gullickson, Miles J. *See* **Dee, Paul E.**

H

Hall, Michael C., and **Selin, Golden.** Spinal Involvement in Gout. A Case Report with Autopsy. 341

Hammack, B. Leslie, and **Enneking, W. F.** Comparative Vascularization of Autogenous and Homogenous-Bone Transplants. 811

Hanakita, Yoshiomi. *See* **Abe, Yasukazu.**

Handler, David. *See* **Dash, Udaya N.**

Harman, Pinckney J.; Magnani, J. L.; Sanwal, S. D.; and **Thompson, W. A. L.** Amino-Acid Chromotography and Transaminase Activity in Denervation Atrophy. . . . 1008

Hassler, William L.; Heyman, Clarence H.; and **Bennett, George W.** Osteochondritis of the Distal Tibial Epiphysis. A Report of Two Cases. 1261

Hatanaka, Ryoshin. *See* **Abe, Yasukazu.**

Heller, Charles J., and **Wiltse, Leon L.** Avascular Necrosis of the Capitellum Humeri (Panner's Disease). A Report of a Case. 513

Henkelmann, Charles. *See* **Huntington, Robert W., Jr.**

Hess, W. E. Giant-Cell Tumor of the Cervical Spine. A Case Report. 480

Heyman, Clarence H. *See* **Hassler, William L.**

Hirohata, Ryozo. *See* **Abe, Yasukazu.**

Hodge, Edwin S. *See* **Ferguson, Albert B., Jr.**

PAGE

Hodgson, A. R., and **Stock, Francis E.** Anterior Spine Fusion for the Treatment of Tuberculosis of the Spine. The Operative Findings and Results of Treatment in the First One Hundred Cases. ... 295
Hoskins, H. Dean. The Versatile Cross-Finger Pedicle Flap. A Report of Twenty-six Cases. ... 261
Houtz, S. J. *See* **Blodgett, William H.**
Howard, Rex J., and **Kleuser, L. P.** Simple Night Splint for Correction of Tibial Torsion in Children. ... 177
Howley, William P. *See* **Rothschild, Eric J.**
Hunter, W. S. *See* **Coleman, S. S.**
Huntington, Robert W., Jr.; Sheffel, Donald J.; Iger, Mortimer; and **Henkelmann, Charles.** Malignant Bone Tumors in Siblings. Ewing's Tumor and an Unusual Tumor Perhaps a Variant of Ewing's Tumor. A Case Report. ... 1065
Hyatt, George W. *See* **Wheeler, Thomas E.**

I

Iger, Mortimer. *See* **Huntington, Robert W., Jr.**
Ikeuchi, Hiroshi. *See* **Bosworth, David M.**
Inglis, Allan. Genetic Implications in Coxa Plana. ... 711
Innis, John J. *See* **Orofino, Caesar.**
Ishizuka, Tadao. *See* **Bosworth, David M.**

J

Jacobs, Bernard, and **Thompson, T. Campbell.** Opposition of the Thumb and Its Restoration. ... 1015
Janes, Joseph M. *See* **Nelson, George E., Jr.**
Johnson, J. T. H., and **Southwick, Wayne O.** Growth Following Transepiphyseal Bone Grafts. An Experimental Study to Explain Continued Growth Following Certain Fusion Operations. ... 1381
Johnson, J. T. H., and **Southwick, Wayne O.** Bone Growth after Spine Fusion. A Clinical Survey. ... 1396
Jones, Malcolm D. Unilateral Laminar Joints of Lumbar Spine. Case Report. ... 1076

K

Kelly, Patrick J. *See* **Nelson, George E., Jr.**
Kennedy, J. C. Arthrodesis of the Ankle with Particular Reference to the Gallie Procedure. A Review of Fifty Cases. ... 1308
King, Don E. *See* **Niebauer, John J.**
Kleuser, L. P. *See* **Howard, Rex J.**
Kosowicz, Jerzy. The Deformity of the Medial Tibial Condyle in Nineteen Cases of Gonadal Dysgenesis. ... 600
Krug, Alfred J. *See* **Schneider, Monroe.**
Kuhlman, Robert E. A Microchemical Study of the Developing Epiphyseal Plate. ... 457

L

Laing, Patrick G. *See* **Ferguson, Albert B., Jr.**
Lansche, W. Edward, and **Ford, Lee T.** Correlation of the Myelogram with Clinical and Operative Findings in Lumbar Disc Lesions. ... 193
Larsen, Loren J. *See* **Bost, Frederic C.**
Larsen, Robert D.; Takagishi, Nahoto; and **Posch, Joseph L.** The Pathogenesis of Dupuytren's Contracture. Experimental and Further Clinical Observations. ... 993
Laurin, C. A. *See* **Farmer, A. W.**
Liebler, William A. *See* **Bosworth, David M.**
Liebler, William A. *See* **Fielding, J. William.**
Lipscomb, Paul R. *See* **Pyka, Rudolf A.**
Lipscomb, Paul R. Duplication of Hypothenar Muscles Simulating Soft-Tissue Tumor of the Hand. Report of a Case. ... 1058
Lowrey, Cedric W. *See* **Orofino, Caesar.**
Lyon, Irving. *See* **Ray, Robert D.**

PAGE

M

MacIntyre, William J. *See* **Cordrey, Lee J.**
McCarroll, H. R., and **Coaten, William.** Attempted Treatment of Scoliosis by Unilateral Vertebral Epiphyseal Arrest. . . . 965
McCaslin, Frank E., Jr. *See* **Sullivan, C. Roger.**
McCauley, John C., Jr. *See* **Rothschild, Eric J.**
McGavran, Malcolm H., and **Spady, Herbert A.** Eosinophilic Granuloma of Bone. A Study of Twenty-eight Cases. . . . 979
McLeay, John F., and **Walske, Benedict R.** Tetracycline Fluorescence in Bone Lesions. Preoperative Tetracycline Administration for the Identification of Abnormal Tissue during Operation and Tetracycline Concentration in Certain Bone Lesions. . . . 940
McMaster, Paul E. Pigmented Villonodular Synovitis with Invasion of Bone. Report of Six Cases. . . . 1170
Magnani, J. L. *See* **Harman, Pinckney J.**
Mangini, Uberto. Flexor Pollicis Longus Muscle. Its Morphology and Clinical Significance. 467
Marmor, Leonard, and **Bechtol, Charles O.** Fracture Separation of the Lower Humeral Epiphysis. Report of a Case. . . . 333
Martin, Albert F. The Pathomechanics of the Knee Joint. I. The Medial Collateral Ligament and Lateral Tibial Plateau Fractures. . . . 13
Martz, Carl D. Talipes Equinus Correction in Cerebral Palsy. . . . 769
Moore, John R. The Closed Fracture of the Long Bones. [Presidential Address. The American Orthopaedic Association.]. . . . 869
Mueller, Martin C., and **Robbins, Jack L.** Intramedullary Lipoma of Bone. Report of a Case. . . . 517

N

Nalebuff, Edward A. *See* **Aitken, Alexander P.**
Nelson, George E., Jr.; Kelly, Patrick J.; Peterson, Lowell F. A.; and **Janes, Joseph M.** Blood Supply of the Human Tibia. . . . 625
Nemethi, Carl E. Reconstruction of the Distal Part of the Thumb after Traumatic Amputation. Restoration of Function and Sensation Using Nerve, Tendon, and Bone from the Amputated Portion. . . . 375
Nicholas, James A.; Wilson, Philip D.; and **Freiberger, Robert.** Pathological Fractures of the Spine. Etiology and Diagnosis. A Review of One Hundred and Five Cases. . . . 127
Nickel, E. D. *See* **Close, J. R.**
Nicoll, E. D. Vere, and **Foster, Eugene A.** Granulomatous Tenosynovitis Due to Beryllium. Histological and Spectrographic Analysis. A Case Report. . . . 1087
Niebauer, John J., and **King, Don E.** Congenital Dislocation of the Knee. . . . 207
Novich, Max M. Adduction Injury of the Knee with Rupture of the Common Peroneal Nerve. Report of a Case. . . . 1372

O

Obletz, Benjamin E. Acute Suppurative Arthritis of the Hip in the Neonatal Period. . . 23
Orofino, Caesar; Innis, John J.; and **Lowrey, Cedric W.** Slipped Capital Femoral Epiphysis in Negroes. A Study of Ninety-five Cases. . . . 1079
Orofino, Caesar; Sherman, Mary S.; and **Schechter, Daniel.** Luschka's Joint—A Degenerative Phenomenon. . . . 853
Oshima, Naozo. *See* **Abe, Yasukazu.**

P

Paletta, Francis X.; Willman, Vallee; and **Ship, Arthur G.** Prolonged Tourniquet Ischemia of Extremities. An Experimental Study on Dogs. . . . 945
Palmer, Rufus. *See* **Cordrey, Lee J.**
Peterson, Lowell F. A. *See* **Nelson, George E., Jr.**
Peterson, Roy R. *See* **Trotter, Mildred.**
Posch, Joseph L. *See* **Larsen, Robert D.**
Pulvertaft, R. Guy. The Treatment of Profundus Division by Free Tendon Graft. . . . 1363
Pyka, Rudolf A., and **Lipscomb, Paul R.** Fractures in Amputees. . . . 499

PAGE

R

Ray, Robert D., and **Lyon, Irving.** Bone Metabolism. IV. The Effect of Radioactive Strontium (Sr^{90}) on the Mobilization of Stable Strontium from the Skeleton of the Rat. 832
Reagan, James W. *See* **Furey, J. George.**
Rhinelander, Frederic W. A Brace Attachment to Permit Knee Mobilization in Hip Spicas. 510
Riordan, Daniel C. The Hand in Leprosy. A Seven-Year Clinical Study. Part I. General Aspects of Leprosy. 661
Riordan, Daniel C. The Hand in Leprosy. A Seven-Year Clinical Study. Part II. Orthopaedic Aspects of Leprosy. 683
Robbins, Jack L. *See* **Mueller, Martin C.**
Rodriguez, Ervin E. *See* **Gartland, John J.**
Rombold, Charles. Depressed Fractures of the Tibial Plateau. Treatment with Rigid Internal Fixation and Early Mobilization. A Preliminary Report. 783
Rose, George G., and **Shindler, Thomas O.** The Cytodifferentiation of Osteoblasts in Tissue Culture. A Description of Cellular Emigrations from Embryo Chick-Leg Bones. 485
Rothschild, Eric J.; **Howley, William P.**; and **McCauley, John C., Jr.** Congenital Recurvatum of the Fibula. 1258
Rubin, Gustav, and **Witten, Morris.** The Talar-Tilt Angle and the Fibular Collateral Ligaments. A Method for the Determination of Talar Tilt. 311
Rubin, Gustav. Letter to the Editor [Correction]. 727
Russe, Otto. Fracture of the Carpal Navicular. Diagnosis, Non-operative Treatment, and Operative Treatment. 759

S

Salter, Robert B., and **Field, Paul.** The Effects of Continuous Compression on Living Articular Cartilage. An Experimental Investigation. 31
Sanwal, S. D. *See* **Harman, Pinckney J.**
Schechter, Daniel. *See* **Orofino, Caesar.**
Schneider, Monroe, and **Krug, Alfred J.** Dislocation of the Hip Secondary to Trochanteric Decubitus. A Complication of Multiple Sclerosis. 1165
Schneider, Richard C. Chronic Neurological Sequelae of Acute Trauma to the Spine and Spinal Cord. Part V. The Syndrome of Acute Central Cervical Spinal-Cord Injury Followed by Chronic Anterior Cervical-Cord Injury (or Compression) Syndrome. 253
Schneider, Richard C. Transposition of the Compressed Spinal Cord in Kyphoscoliotic Patients with Neurological Deficit. With Special Reference to the Vascular Supply of the Cord. 1027
Schottstaedt, Edwin R. *See* **Bost, Frederic C.**
Schottstaedt, Edwin R. *See* **Sutherland, David H.**
Schwarz, Gerhart S. Late Appearance and Evolution of a Cortical Defect in a Boy with Delayed Puberty. 173
Sedlin, Elias D. A Device for Stress Inversion or Eversion Roentgenograms of the Ankle. 1184
Selin, Golden. *See* **Hall, Michael C.**
Sheffel, Donald J. *See* **Huntington, Robert W., Jr.**
Sherman, Mary S. *See* **Orofino, Caesar.**
Shindler, Thomas O. *See* **Rose, George G.**
Ship, Arthur G. *See* **Paletta, Francis X.**
Shroyer, Russell N. Ossification of the Posterior Aspect of the Elbow. Two Cases. 1156
Shroyer, Russell N.; **Fortson, Charles H.**; and **Theodotou, C. B.** Delayed Neurological Sequelae of a Retained Foreign Body (Lead Bullet) in the Intervertebral Disc Space. 595
Skovron, Michael. *See* **Gartland, John J.**
Smith, Alan DeForest. Osteochondritis of the Knee Joint. A Report of Three Cases in One Family and a Discussion of the Etiology and Treatment. 289
Smith, Albert G.; **Coonrad, Ralph W.**; **Boyer, Delos W.**; and **Bugg, Everett I., Jr.** Unusual Case of Multiple Abnormalities of Bone with Malignant Degeneration. 1329
Smith, James W. *See* **Boyes, Joseph H.**
Smith, Lyman. Deformity Following Supracondylar Fractures of the Humerus. 235
Smith, R. Glenn. *See* **Dee, Paul E.**
Smith, Richard L. *See* **Altman, Donald H.**
Sofield, Harold A. Growth of the Vineyards [Presidential Address, The American Academy of Orthopaedic Surgeons].

PAGE

Southwick, Wayne O. *See* **Johnson, J. T. H.**
Spady, Herbert A. *See* **McGavran, Malcolm H.**
Staples, O. Sherwin. Injuries to the Medial Ligaments of the Ankle. Result Study. 1287
Stock, Francis E. *See* **Hodgson, A. R.**
Sullivan, C. Roger, and **McCaslin, Frank E., Jr.** Further Studies on Experimental Spondylitis and Intercorporeal Fusion of the Spine 1339
Sutherland, David H.; Bost, Frederic C.; and **Schottstaedt, Edwin R.** Electromyographic Study of Transplanted Muscles about the Knee in Poliomyelitic Patients..... 919
Swanson, Alfred B. Surgery of the Hand in Cerebral Palsy and the Swan-Neck Deformity. 951

T

Takagishi, Nahoto. *See* **Larsen, Robert D.**
Tambakis, A. *See* **Fielding, J. William.**
Teng, Paul. Spondylosis of the Cervical Spine with Compression of the Spinal Cord and Nerve Roots. 392
Theodotou, C. B. *See* **Shroyer, Russell N.**
Thompson, T. Campbell. *See* **Jacobs, Bernard.**
Thompson, W. A. L. *See* **Harman, Pinckney J.**
Todd, F. N. *See* **Close, J. R.**
Trotter, Mildred; Broman, George E.; and **Peterson, Roy R.** Densities of Bones of White and Negro Skeletons. 50
Troup, James B., and **Bickel, William H.** Malignant Disease of the Extremities Treated by Exarticulation. Analysis of Two Hundred and Sixty-four Consecutive Cases with Survival Rates. 1041

V

Vasilas, Anthony; Grieco, R. Vincent; and **Bartone, Noel F.** Roentgen Aspects of Injuries to the Pisiform Bone and Pisotriquetral Joint. 1317
Verdan, Claude E. Primary Repair of Flexor Tendons. 647

W

Waisbren, Burton A. Pyogenic Osteomyelitis and Arthritis of the Spine Treated with Combinations of Antibiotics and Gamma Globulin. A Preliminary Report. 414
Walske, Benedict R. *See* **McCleay, John F.**
Weinfeld, Marvin S. *See* **Cohen, Jonathan.**
Wheeler, Thomas E., and **Hyatt, George W.** A Study of the Residual Nitrogen in Bone Following Extraction with Ethylenediamine. 1435
Whitesides, Thomas E., Jr., and **Ackerman, Lauren V.** Desmoplastic Fibroma. A Report of Three Cases. 1143
Willman, Vallee. *See* **Paletta, Francis X.**
Wilson, James N. *See* **Boyes, Joseph H.**
Wilson, John T. *See* **Flynn, J. Edward.**
Wilson, Philip D. *See* **Nicholas, James A.**
Wiltse, Leon L. *See* **Heller, Charles J.**
Winters, Jack L., and **Cahen, Irvin.** Acute Hematogenous Osteomyelitis. A Review of Sixty-six Cases. 691
Witten, Morris. *See* **Rubin, Gustav.**
Woolfrey, B. F., and **Chandler, E. F.** Manifestations of Osgood-Schlatter's Disease in Late Teen Age and Early Adulthood. 327

Z

Zancolli, Eduardo. Transplantation of the Index Finger in Congenital Absence of the Thumb. 658

SUBJECT INDEX

VOLUME 42-A

A

PAGE

Abduction Fracture of the Femoral Neck ... 471
Abscess, Paravertebral ... 295
Abt-Letterer-Siwe Syndrome ... 979
Accessory Head of Flexor Pollicis Longus ... 467
Achilles-Tendon Lengthening ... 769
Acidosis ... 437
Action-Potential Counts, Motor-Unit. Their Significance in Isometric and Isotonic Contractions. J. R. Close, E. D. Nickel, and F. N. Todd ... 1207
Action-Potential Patterns of Leg Muscles ... 59, 919, 1207
Acute Hematogenous Osteomyelitis. A Review of Sixty-six Cases. Jack L. Winters and Irvin Cahen ... 691
Acute Suppurative Arthritis of the Hip in the Neonatal Period. Benjamin E. Obletz ... 23
Adduction Injury of the Knee with Rupture of the Common Peroneal Nerve. Report of a Case. Max M. Novich ... 1372
Adductor Myotomy and Obturator Neurectomy for the Correction of Adduction Contracture of the Hip in Cerebral Palsy. Henry H. Banks and William T. Green ... 111
Adenocarcinoma, Radical Mastectomy for, A Rare Subperiosteal Metastasis Eroding the Femoral Cortex Eight Years after. Charles A. Borgia ... 1084
Alabama Orthopaedic Society ... 730
Alkaptonuria from One Family Tree with Special Reference to Osteo-Arthrosis. Alkaptonurica, Thirteen Cases of. Yasukazu Abe, Naozo Oshima, Ryoshin Hatanaka [correctly, Yoshiomi Hanakita], Tamikazu Amako, and Ryozo Hirohata ... 817
Alloys for Metal Implants ... 77
American Academy for Cerebral Palsy ... 1283
American Academy of Orthopaedic Surgeons, The ... 1282
American Academy of Orthopaedic Surgeons, The [Presidential Address.] Growth of the Vineyards. Harold A. Sofield ... 344
American Association for Automotive Medicine ... 1109
American Board of Orthopaedic Surgery ... 562, 731, 1109, 1283
American Orthopaedic Association, The ... 365, 560
American Orthopaedic Association, The. The Exchange Fellowship Committee ... 365, 562, 731, 1109, 1283
American Orthopaedic Association, The. [Presidential Address.] The Closed Fracture of the Long Bones. John R. Moore ... 869
American Society for Surgery of the Hand ... 1282
Amino-Acid Chromatography and Transaminase Activity in Denervation Atrophy. Pinckney J. Harman, J. L. Magnani, S. D. Sanwal, and W. A. L. Thompson ... 1008
Amputation ... 1041
Amputation of Finger ... 261
Amputation, Syme ... 1
Amputation, Traumatic, of Thumb 375
Amputees, Fractures in. Rudolf A. Pyka and Paul R. Lipscomb ... 499
Aneurysmal Bone Cyst ... 480
Angulation of Vertebrae ... 1413
Ankle, Arthrodesis of the, with Particular Reference to the Gallie Procedure. A Review of Fifty Cases. J. C. Kennedy ... 1308
Ankle Assembly, Prosthetic ... 226
Ankle, A Device for Stress Inversion or Eversion Roentgenograms of the. Elias D. Sedlin ... 1184
Ankle, Injuries to the Medial Ligaments of the. Result Study. O. Sherwin Staples ... 1287
Ankle Mortise ... 311, 1287
Ankle Sprains ... 311
Ankylosing Spondylitis ... 127
Ankylosis of the Elbow ... 1156
Anomalies, Congenital ... 1
Anorganic Bone ... 1435
Anterior Spine Fusion for the Treatment of Tuberculosis of the Spine. The Operative Findings and Results of Treatment in the First One Hundred Cases. A. R. Hodgson and Francis E. Stock ... 295
Antibiotics in Treatment of Osteomyelitis ... 414, 429, 691
Apatite Lattice ... 437
Apical Lung Carcinoma, The Orthopaedist and. Paul E. Dee, R. Glenn Smith, Miles J. Gullickson, and Charles S. Ballinger ... 605
Aponeurosis, Tearing of ... 993
Apparatus
A Brace Attachment to Permit Knee Mobilization in Hip Spicas. Frederic W. Rhinelander ... 510
A Device for Stress Inversion or Eversion Roentgenograms of the Ankle. Elias D. Sedlin ... 1184

PAGE

Simple Night Splint for Correction of Tibial Torsion in Children. Rex J. Howard and L. P. Kleuser 177
Arachnoiditis 392
Archipterygium Theory of Gegenbauer 1
Arterial Supply of Spinal Cord 1027
Arthritis, Acute Suppurative, of the Hip in the Neonatal Period. Benjamin E. Obletz 23
Arthritis, Cervical Hypertrophic 253
Arthritis, Degenerative 853
Arthritis, Pathogenesis of Degenerative 31
Arthritis of Spine 414
Arthritis, Suppurative 1223
Arthrodesis of the Ankle with Particular Reference to the Gallie Procedure. A Review of Fifty Cases. J. C. Kennedy 1308
Arthrosis after Fracture of Navicular 759
Articular Cartilage 31
Aseptic Necrosis in Children after Traumatic Dislocation of the Hip 705
Association of Bone and Joint Surgeons 365
Atlantic Provinces Orthopaedic Society 730
Atlanto-Axial Joint with Dislocation, Congenital Malformation of the. Earl W. Brannon 1377
Atrophy, Denervation 1008
Atrophy of Skeletal Muscle 278, 617
Attempted Treatment of Scoliosis by Unilateral Vertebral Epiphyseal Arrest. H. R. McCarroll and William Costen 965
Autogenous-Tendon Transplants 91
Avascular Necrosis 471
Avascular Necrosis of the Capitellum Humeri (Panner's Disease). A Report of a Case. Charles J. Heller and Leon L. Wiltse 513

B

Bacteremia, Transient 1191
Badgley, Carl E. 1283
Beryllium, Granulomatous Tenosynovitis 1087
Blood Supply of the Human Tibia. George E. Nelson, Jr., Patrick J. Kelly, Lowell F. A. Peterson, and Joseph M. Janes 625
Blood Supply of Intervertebral Disc 1191
Blount's Disease (Tibia Vara) 600
Bone Absorption in Leprosy 683
Bone Alterations in Experimental Immobilization 737
Bone Atrophy 1349
Bone, Chondrosarcoma of 1239
Bone Composition in Acidotic Rats 437
Bone Cysts, Simple. Studies of Cyst Fluid in Six Cases with a Theory of Pathogenesis. Jonathan Cohen 609
Bone Demineralization 127
Bone Disease, Bizarre 1329

PAGE

Bone, Endochondral 457
Bone, Eosinophilic Granuloma of. A Study of Twenty-eight Cases. Malcolm H. McGavran and Herbert A. Spady 979
Bone Explants 485
Bone Feathering 447
Bone Grafts, Transepiphyseal, Growth Following. An Experimental Study to Explain Continued Growth Following Certain Fusion Operations. J. T. H. Johnson and Wayne O. Southwick 1381
Bone Growth after Spine Fusion. A Clinical Survey. J. T. H. Johnson and Wayne O. Southwick 1396
Bone Infarcts, Fibrosarcoma Arising at the Site of. A Report of Two Cases. J. George Furey, Miguel Ferrer-Torells, and James W. Reagan 802
Bone, Injuries to the Pisiform 1317
Bone Invasion in Pigmented Villonodular Synovitis 1170
Bone, Intramedullary Lipoma of 517
Bone Lesions, Tetracycline Fluorescence in. Preoperative Tetracycline Administration for the Identification of Abnormal Tissue during Operation and Tetracycline Concentration in Certain Lesions. John F. McLeay and Benedict R. Walske 940
Bone Lesions, Traumatically Induced, in Infants 407
Bone Magnesium Content in Normal and Acidotic Rats. William H. Bergstrom and Ellen H. Bell 437
Bone Marrow, Depression of 1239
Bone Metabolism 1349
Bone Metabolism. IV. The Effect of Radioactive Strontium (Sr^{90}) on the Mobilization of Stable Strontium from the Skeleton of the Rat. Robert D. Ray and Irving Lyon 832
Bone, Micropetrotic 144
Bone Physiology 138
Bone Repair, Plastics and [Editorial] 521
Bone, A Study of the Residual Nitrogen in, Following Extraction with Ethylenediamine. Thomas E. Wheeler and George W. Hyatt 1435
Bone Transplants, Vascularization of 811
Bone Tumor of Fibroblastic Origin 1143
Bone Tumors, Malignant, in Siblings 1065
Bone, Well Differentiated Fibrosarcoma of 1151
Bones, Densities of, of White and Negro Skeletons. Mildred Trotter, George E. Broman, and Roy R. Peterson 50
Bones, Long, Closed Fracture of the 869
Book Reviews
Albert, F., and Medawar, P. B. Biological Problems of Grafting. A Symposium 735
Anderson, Miles H.; Bechtol, Charles O.; and Sollars, Raymond E. A Handbook of Clinical Practices Related to Artificial Limbs. Clinical

PAGE

Prosthetics for Physicians and Therapists ... 734
Breig, Alf. Biomechanics of the Central Nervous System. Some Basic Normal and Pathologic Phenomena ... 1285
Brookes, R. Barrie. Re-education of the Injured Shoulder ... 1284
Byrne, John J. The Hand: Its Anatomy and Diseases ... 191
Davis, Loyal [Editor]. Christopher's Text Book of Surgery. Ed. 7 ... 1454
Eckardt, Robert E. Industrial Carcinogens ... 372
Gardner, Ernest; Gray, Donald J.; and O'Rahilly, Ronan. Anatomy: A Regional Study of Human Structure ... 1453
Goldstein, Louis A. Surgical Treatment of Scoliosis. With an Appendix "Anesthesia in Scoliosis", by D. Vernon Thomas ... 733
Hanraets, P. R. M. J. The Degenerative Back and Its Differential Diagnosis ... 735
Hardy, James D. Total Surgical Management ... 371
Iselin, M.; Gosse, L.; Boussard, S.; and Benoist, D. Atlas de Technique Opératoire. Chirurgie de la Main ... 1453
Last, R. J. Anatomy: Regional and Applied ... 735
Licht, Sidney [Editor]. Therapeutic Electricity and Ultraviolet Radiation ... 370
Lowman, Charles LeRoy, and Young, Carl Haven. Postural Fitness. Significance and Variances ... 1284
McGregor, Ian A. Fundamental Techniques of Plastic Surgery and Their Surgical Applications ... 1284
Markowitz, J.; Archibald, J.; and Downie, H. G. Experimental Surgery. Including Surgical Physiology. Ed. 4 ... 373
Matzen, Peter E., Lehrbuch der Orthopädie ... 1455
Moore, Francis D. Metabolic Care of the Surgical Patient ... 1454
Moss, William T. Therapeutic Radiology ... 371
Paul, Lester W., and Juhl, John J. The Essentials of Roentgen Interpretation ... 192
Poppen, James L. An Atlas of Neurosurgical Techniques ... 1455
Ramsey, George H. S.; Tristan, Theodore A.; Watson, James S., Jr.; Weinberg, Sydney; and Cornwell, William S. [Editors]. Cinefluorography: Proceedings of the First Annual Symposium on Cinefluorography ... 1454
Reynolds, Fred C. [Editor]. Instructional Course Lectures. Volume XVI. The American Academy of Orthopaedic Surgeons ... 734
Rodahl, Kaare; Nicholson, Jesse T.; and Brown, Ernest M. [Editors]. Bone as a Tissue ... 564

PAGE

Salmon, F. Cuthbert, and Salmon, Christine F. Rehabilitation Center Planning ... 372
Schwartz, Laszlo. Disorders of the Temporomandibular Joint. Diagnosis, Management, Relations to Occlusion of Teeth ... 373
Steindler, Arthur. Lectures on Interpretation of Pain in Orthopedic Practice ... 564
Stowens, Daniel. Pediatric Pathology ... 734
Trifau, André; Bureau, Henri; and Payan, Henri. Tumeurs Bénignes des Os et Dystrophies Pseudo-tumorales ... 370
Turek, Samuel L. Orthopaedics: Principles and Their Application ... 191
Weiss, Thomas E., and Segaloff, Albert. Gouty Arthritis and Gout. An Ancient Disease with Modern Interest ... 733
Whyte, G. N. Principles of Radiation Dosimetry ... 371
Wiles, Philip. Essentials of Orthopaedics. Ed. 3 ... 736
Bovine-Tendon Transplants ... 91
Bowing, Posterior, of the Fibula ... 1258
Brace Attachment to Permit Knee Mobilization in Hip Spicas, A. Frederic W. Rhinelander ... 510
Bracing in Cerebral Palsy ... 769
Bursitis, Pigmented Villonodular ... 1170

C

Caisson Disease ... 820
Calcaneofibular Ligament ... 311
Calcaneus, Well Differentiated Fibrosarcoma of the ... 1151
Capitate ... 1051
Capitellum Humeri (Panner's Disease), Avascular Necrosis of the. A Report of a Case. Charles J. Heller and Leon L. Wiltse ... 513
Capitellum, Ossification Center of ... 333
Carbohydrate Metabolism ... 457
Carcinoma, Apical Lung ... 605
Carpal Navicular, Fracture of ... 759
Carpometacarpal Joint, First ... 1111
Carpus, Volar Transnavicular Perilunar Dislocation of the. Alexander P. Aitken and Edward A. Nalebuff ... 1051
Cartilage Alterations ... 737
Cartilage, The Effects of Continuous Compression on Living Articular. An Experimental Investigation. Robert B. Salter and Paul Field ... 31
Cartilage Retention of Radioactive Sulphur ... 1239
Cartilage, Sulphate Metabolism in. II. S^{35}-Sulphate Uptake and Total Sulphate in Cartilage Slices. Rose Robyns Coelho and O. Donald Chrisman ... 165
Case of Compression of Subclavian Vessels by a Fractured Clavicle Treated by Excision of the First Rib, A. Udaya N. Dash and David Handler ... 798

PAGE

Cauda Equina Compression 595
Cellular Emigrations 485
Cerebral Palsy 111, 769, 859, 951, 1223
Cerebral Palsy, Talipes Equinus Correction in. Carl D. Martz 769
Cervical Spinal-Cord Injury 253
Cervical Spine, Giant-Cell Tumor of the. A Case Report. W. E. Hess 480
Cervical Spine, Spondylosis of the, with Compression of the Spinal Cord and Nerve Roots. Paul Teng .. 392
Cervical Spine, Stabilization of the, by Anterior Fusion. R. W. Bailey and C. E. Badgley 565
Cervical Vertebrae 853
Cervical Vertebral Bodies, Destructive Lesions of 565
Charcot Joints in Leprosy 683
Charcot-Marie-Tooth Disease 661
Chemical Determination of Cyst Fluid 609
Chondrosarcoma 1041
Chondrosarcomata, Radioactive Sulphur in. The Distribution, Concentration, and Effects of S^{35} after Intravenous Administration to Patients with Chondrosarcoma. Raymond G. Gottschalk 1239
Chromatography, Amino-Acid 1008
Chrondroitin Sulphate 165
Chronic Neurological Sequelae of Acute Trauma to the Spine and Spinal Cord. Part V. The Syndrome of Acute Central Cervical Spinal-Cord Injury Followed by Chronic Anterior Cervical-Cord Injury (or Compression) Syndrome. Richard C. Schneider 253
Clavicle, Congenital Pseudarthrosis of the. Report of a Case with Resection and Bone-Grafting. Earnest B. Carpenter and Roland G. Garrett 337
Clavicle, Fractured, Treated by Excision of the First Rib, A Case of Compression of Subclavian Vessels by a. Udaya N. Dash and David Handler 798
Clinical and Electromyographic Evaluation of Patients with Anterior Transposition of Peroneal Tendons. William H. Blodgett and S. J. Houtz .. 59
Clinical Experiences with the S.A.C.H. Foot Prosthesis. Everett J. Gordon and Joseph Ardizzone 226
Clinical Orthopaedic Society 368
Closed Fracture of the Long Bones, The. [Presidential Address, The American Orthopaedic Association.] John R. Moore 869
Club-Foot 151
Cobalt Alloys 77
Comparative Vascularization of Autogenous and Homogenous-Bone Transplants. B. Leslie Hammack and W. F. Enneking 811
Compression of Spinal Cord 253, 1027
Compression of Spinal Cord and Nerve Roots 392
Compression of Subclavian Vessels .. 798
Congenital Absence of the Fibula. A. W. Farmer and C. A. Laurin 1
Congenital Dislocation of the Hip ... 1223
Congenital Dislocation of the Knee. John J. Niebauer and Don E. King ... 207
Congenital Malformation of the Atlanto-Axial Joint with Dislocation. Earl W. Brannon 1377
Congenital Pseudarthrosis of the Clavicle. Report of a Case with Resection and Bone-Grafting. Earnest B. Carpenter and Roland G. Garrett .. 337
Congenital Recurvatum of the Fibula. Report of One Case. Eric J. Rothschild, William P. Howley, and John C. McCauley, Jr. 1258
Contracture, External Rotation, of Thumb 1015
Contracture, Dupuytren's 993
Conservative Treatment of Impacted Fractures of the Femoral Neck. A Report of Fifty Cases. Henry B. Crawford 471
[Correction.] Letter to the Editor. Gustav Rubin 727
Correlation of the Myelogram with Clinical and Operative Findings in Lumbar Disc Lesions. W. Edward Lansche and Lee T. Ford 193
Corrosion 77
Corrosion of a Steinmann Pin Used for Intramedullary Fixation, Failure by. Jonathan Cohen and W. Sanford Foultz 1201
Cortical Defect in a Boy with Delayed Puberty, Late Appearance and Evolution of a. Gerhart S. Schwarz 173
Coxa Magna 1223
Coxa Plana, Genetic Implications in. Allan Inglis 711
Coxa Plana in Monozygotic Male Twins. Adolphus W. Dunn 178
Coxa Valga 111
Cross-Finger Pedicle Flap 261
Cruciate Ligaments 13
Cubitus Varus 235
Curve, The Problem of the Primary. John R. Cobb 1413
Cyst Fluid, Study of Bone 609
Cystlike Rarefactions, Transitory ... 173
Cytodifferentiation of Osteoblasts in Tissue Culture, The. A Description of Cellular Emigrations from Embryo Chick-Leg Bones. George G. Rose and Thomas O. Shindler 485
Czechoslovak Association of Plastic Surgeons 368

D

Decubitus Ulcer 1165
Deformity Following Supracondylar Fractures of the Humerus. Lyman Smith 235
Deformity of the Medial Tibial Con-

PAGE

dyle in Nineteen Cases of Gonodal Dysgenesis, The. Jerzy Kosowicz.... 600
Delayed Neurological Sequelae of a Retained Foreign Body (Lead Bullet) in the Intervertebral Disc Space. Russell N. Shroyer, Charles H. Fortson, and C. B. Theodotou..... 595
Deltoid Ligament of the Ankle..... 1287
Denervation Atrophy, Amino-Acid Chromatography and Transaminase Activity in. Pinckney J. Harman, J. L. Magnani, S. D. Sanwal, and W. A. L. Thompson..... 1008
Denervation of Gastrocnemius..... 769
Densities of Bones of White and Negro Skeletons. Mildred Trotter, George E. Broman, and Roy R. Peterson..... 50
Depressed Fractures of the Tibial Plateau. Treatment with Rigid Internal Fixation and Early Mobilization. A Preliminary Report. Charles Rombold..... 783
Desmoplastic Fibroma. A Report of Three Cases. Thomas E. Whitesides, Jr., and Lauren V. Ackerman..... 1143
Device for Stress Inversion or Eversion Roentgenograms of the Ankle, A. Elias D. Sedlin..... 1184
Diaphyseal Aclasis..... 600
Digital Sheath, Repair of Tendons in the..... 647
Digital Theca..... 1363
Digital Transplantation..... 658
Disc, Disruption of Intervertebral... 565
Disc, Lesion of the Intervertebral, in Children..... 1191
Disc Lesions..... 193
Dislocation of the Atlanto-Axial Joint 1377
Dislocation of the Hip..... 111
Dislocation of the Hip Secondary to Trochanteric Decubitus. A Complication of Multiple Sclerosis. Monroe Schneider and Alfred J. Krug..... 1165
Dislocation of the Hip, Traumatic, in Children..... 705
Dorsiflexion Reflex of the Foot..... 859
Duplication of Hypothenar Muscles Simulating Soft-Tissue Tumor of the Hand. Report of a Case. Paul R. Lipscomb..... 1058
Dupuytren's Contracture, The Pathogenesis of. Experimental and Further Clinical Observations. Robert D. Larsen, Nahoto Takagishi, and Joseph L. Posch..... 993
Dysplasia of Limbs..... 1
Dystrophic Muscles..... 1207

E

[Editorial.] Plastics and Bone Repair 521
Effects of Continuous Compression on Living Articular Cartilage, The. An Experimental Investigation. Robert B. Salter and Paul Field..... 31

PAGE

Effect of a Tibial-Tubercle Transplant in Children on the Growth of the Upper Tibial Epiphysis, The. J. William Fielding, William A. Liebler, and A. Tambakis..... 1426
Elbow..... 235, 333, 513
Elbow, Ossification of the Posterior Aspect of the. Two Cases. Russell N. Shroyer..... 1156
Electrical Response of Human Skeletal Muscle to Passive Stretch, The. Robert O. Becker..... 1091
Electromyographic Study of Transplanted Muscles about the Knee in Poliomyelitic Patients. David H. Sutherland, Frederic C. Bost, and Edwin R. Schottstaedt..... 919
Electromyography..... 59, 919, 1091, 1207
Endochondral Ossification..... 457
Endosteal Bone..... 485
Enzymatic Characteristics of Endochondral Bone..... 457
Enzyme, Cyanide Sensitive..... 165
Eosinophilic Granuloma of Bone. A Study of Twenty-eight Cases. Malcolm H. McGavran and Herbert A. Spady..... 979
Epiphyseal Arrest..... 965
Epiphyseal Cartilage, Destruction of 1239
Epiphyseal-Metaphyseal Vascular System..... 626
Epiphyseal Plate, A Microchemical Study of the Developing. Robert E. Kuhlman..... 457
Epiphyses, Bone Grafts across..... 1381
Epiphyses, Deformity of Femoral and Tibial..... 207
Epiphysis, Femoral, Experimental Trauma to..... 439
Epiphysis, Fracture Separation of the Lower Humeral. Report of a Case. Leonard Marmor and Charles O. Bechtol..... 333
Epiphysis, Osteochondrosis of the Distal Tibial..... 1261
Epiphysis, Slipped Capital Femoral, in Negroes. A Study of Ninety-five Cases. Caesar Orofino, John J. Innis, and Cedric W. Lowrey..... 1079
Epiphysis, Upper Tibial..... 1426
Epiphysitis of Femoral Head..... 1223
Equinovalgus Deformity..... 1119
Equinovarus, Talipes..... 151
Equinus Deformity in Cerebral Palsy 769
Ethylenediamine Extraction..... 1435
Etiology of Osteochondritis Dissecans 289
Eversion Reflex of the Foot..... 859
Ewing's Tumor..... 1041
Ewing's Tumor and an Unusual Tumor Perhaps a Variant of Ewing's Tumor. A Case Report. Malignant Bone Tumors in Siblings. Robert W. Huntington, Jr., Donald J. Sheffel, Mortimer Iger, and Charles Henkelmann..... 1065
Exarticulation, Malignant Disease of the Extremities Treated by. Analysis of Two Hundred and Sixty-four

PAGE

Consecutive Cases with Survival Rates. James B. Troup and William H. Bickel ... 1041
Excerpta Medica Foundation ... 562
Exchange Fellows (British) ... 365
Exchange Fellows, 1960 [Photograph] 1104
Experimental Excision of Muscles in the Weanling Rat. Jonathan Cohen and Marvin S. Weinfeld ... 278
Experimental Immobilization and Remobilization of Rat Knee Joints. [Correction, page 1108.] E. Burke Evans, G. W. N. Eggers, James K. Butler, and Johanna Blumel ... 737
Extra-Articular Hip Arthrodesis. Salvage Procedure for Persistent Pain Following Replacement Arthroplasty: A Case Report. S. S. Coleman and W. S. Hunter ... 1062

F

Failure by Corrosion of a Steinmann Pin Used for Intramedullary Fixation. Jonathan Cohen and W. Sanford Foultz ... 1201
Feathering, A New Bone Affection. Harold M. Frost ... 447
Femoral Condyle, Medial, Osteochondritis of ... 289
Femoral Cortex, Erosion of, by Subperiosteal Metastasis ... 1084
Femoral Epiphysis in Rabbits—III, A Study of Experimental Trauma and Attempts to Stimulate Growth of the Lower. Lee T. Ford and Gregorio M. Canales ... 439
Femoral Epiphysis, Slipping of, in Negroes ... 1079
Femoral Head to Acetabulum Relationship ... 1223
Femoral Head, Osteochondritis Dissecans of ... 777
Femoral Neck, Impacted Fractures of the ... 471
Femur, Osteomyelitis of ... 23
Fibroma, Desmoplastic. A Report of Three Cases. Thomas E. Whitesides, Jr., and Lauren V. Ackerman ... 1143
Fibrosarcoma ... 1041
Fibrosarcoma Arising at the Site of Bone Infarcts. A Report of Two Cases. J. George Furey, Miguel Ferrer-Torells, and James W. Reagan ... 802
Fibrosarcoma, Well Differentiated, of the Calcaneus. Report of a Case Treated by Resection. Raphael R. Goldenberg ... 1151
Fibula, Congenital Absence of the. A. W. Farmer and C. A. Laurin ... 1
Fibula, Congenital Recurvatum of the. Report of One Case. Eric J. Rothschild, William P. Howley, and John C. McCauley, Jr. ... 1258
Fibula, Fracture of ... 1287
Fibular Collateral Ligaments, The Talar-Tilt Angle and the. A Method for the Determination of Talar Tilt. [Correction, page 727.] Gustav Rubin and Morris Witten ... 311
Fifth International Poliomyelitis Conference ... 731
Finger, Amputation of ... 261
Finger, Index, Transplantation of the, in Congenital Absence of the Thumb. Eduardo Zancolli ... 658
Finger, Pedicle Flap ... 261
Fingers, Extension Deformity of ... 951
Fingers, Flexion Deformity of ... 951
Flexor Digitorum Profundus ... 467
Flexor Pollicis Longus Muscle. Its Morphology and Clinical Significance. Uberto Mangini ... 467
Flexor-Tendon Ruptures in the Forearm and Hand. Joseph H. Boyes, James N. Wilson, and James W. Smith 637
Flexor Tendons, Primary Repair of. Claude E. Verdan ... 647
Flexor Tendons, Replacement of Divided ... 1363
Fluorescence, Tetracycline ... 940
Foot, Deformities of ... 1
Foot, Paralytic Valgus ... 1119
Foot Prosthesis (S.A.C.H.) ... 226
Foot, Release of Ligamentous Tissues of the ... 151
Foot, Tonic Reflexes of the. Their Orthopaedic Significance in Normal Children and in Children with Cerebral Palsy. William R. Duncan ... 859
Foreign Body Retained in Intervertebral Disc Space ... 595
Fracture of the Carpal Navicular. Diagnosis, Non-operative Treatment, and Operative Treatment. Otto Russe ... 759
Fracture of Clavicle ... 798
Fracture, Closed, of the Long Bones ... 869
Fracture-Dislocation of Femoral Epiphysis ... 407
Fracture-Dislocations, Cervical Spine 565
Fracture Separation of the Lower Humeral Epiphysis. Report of a Case. Leonard Marmor and Charles O. Bechtol ... 333
Fractures in Amputees. Rudolf A. Pyka and Paul R. Lipscomb ... 499
Fractures of the Femoral Neck, Conservative Treatment of Impacted. A Report of Fifty Cases. Henry B. Crawford ... 471
Fractures of the Greater Multangular 1111
Fractures of Lateral Tibial Plateau ... 13
Fractures, Pathological, of the Spine 127
Fractures, Stress, in Spine Fusions ... 1396
Fractures, Supracondylar, of the Humerus ... 235
Fractures of the Tibial Plateau ... 783
Further Studies on Experimental Spondylitis and Intercorporeal Fusion of the Spine. C. Roger Sullivan and Frank E. McCaslin, Jr. ... 1339
Fusion, Anterior Spine ... 295
Fusion, Gallie Subtalar ... 1308

PAGE

Fusion, Interbody, of the Spine 1339
Fusion, Spontaneous Interbody 429

G

Gait 59, 919
Gait, after Ankle Fusion 1308
Gallie Procedure 1308
Gamma Globulin, Pyogenic Osteomyelitis and Arthritis of the Spine Treated with Combinations of Antibiotics and. A Preliminary Report. Burton A. Waisbren 414
Gantzer's Muscle 467
Gastrocnemius Recession 769
Genetic Implications in Coxa Plana. Allan Inglis 711
Genu Recurvatum 207
Giant-Cell Tumor of the Cervical Spine. A Case Report. W. E. Hess 480
Glucose-6-Phosphate 457
Gluteus Maximus, Phase Activity of 919
Gonadal Dysgenesis 600
Gordon Research Conferences, The 366
Gout, Spinal Involvement in. A Case Report with Autopsy. Michael C. Hall and Golden Selin 341
[Graft.] Cross-Finger Pedicle Flap 261
Granuloma, Eosinophilic 979
Granulatomous Tenosynovitis Due to Beryllium. Histological and Spectrographic Analysis. A Case Report. K. D. Vere Nicoll and Eugene A. Foster 1087
Grice, David S. 1282
Growth of Bone after Spine Fusion 1396
Growth Disturbance after Elbow Fracture 235
Growth Following Certain Fusion Operations 1381
Growth Following Transepiphyseal Bone Grafts. An Experimental Study to Explain Continued Growth Following Certain Fusion Operations. J. T. H. Johnson and Wayne O. Southwick 1381
Growth Stimulation, Experimental 439
Growth of the Upper Tibial Epiphysis 1426
Growth of the Vineyards [Presidential Address, The American Academy of Orthopaedic Surgeons]. Harold A. Sofield 344
Guillotine Amputation of Fingers 261
Gunstock Deformity 235

H

Halisteresis 447
Hamstring Activity, Conversion of 919
Hanakita, Yoshiomi 1283
Hand, Duplication of Hypothenar Muscles of the 1058
Hand, Flexor-Tendon Ruptures in the Forearm and 637
Hand Function and Opposition of the Thumb 1015

PAGE

Hand in Leprosy, The. A Seven-Year Clinical Study. Part I. General Aspects of Leprosy. Daniel C. Riordan 661
Hand in Leprosy, The. A Seven-Year Clinical Study. Part II. Orthopaedic Aspects of Leprosy. Daniel C. Riordan 683
Hand-Schüller-Christian Syndrome 979
Hand, Surgery of the, in Cerebral Palsy and the Swan-Neck Deformity. Alfred B. Swanson 951
Hansen's Disease 661
Haversian Canals 626
Haversian Systems 138
Healing of Autogenous-Tendon Transplants 91
Heel-Cord Contracture 59
Hematogenous Osteomyelitis 691
Hematogenous Pyogenic Vertebral Osteomyelitis. Alexander Garcia, Jr., and S. Ashby Grantham 429
Hemosiderin Deposits 993
Heparin 945
Heterogenous and Autogenous-Tendon Transplants. An Experimental Study of Preserved Bovine-Tendon Transplants in Dogs and Autogenous-Tendon Transplants in Dogs. J. Edward Flynn, John T. Wilson, Charles G. Child, III, and James H. Graham 91
Heterotopic Ossification of the Elbow 1156
Hindquarter Amputation 1041
Hip, Adduction Contracture of 111
Hip Arthrodesis, Extra-Articular. Salvage Procedure for Persistent Pain Following Replacement Arthroplasty: A Case Report. S. S. Coleman and W. S. Hunter 1062
Hip, Congenital Dislocation of 207
[Hip] Coxa Plana in Monozygotic Male Twins. Adolphus W. Dunn 178
Hip, Dislocation of 111
Hip, Dislocation of the, Secondary to Trochanteric Decubitus. A Complication of Multiple Sclerosis. Monroe Schneider and Alfred J. Krug 1165
[Hip] Genetic Implications in Coxa Plana. Allan Inglis 711
Hip-Joint Disarticulation 1041
Hip Joint, Traumatic Dislocation of the, in Children. A Report by the Scientific Research Committee of the Pennsylvania Orthopedic Society. John J. Gartland, *Chairman*, Albert B. Ferguson, Jr., William F. Donaldson, Marvin C. Goodman, Ervin E. Rodriguez, and Michael Skovron 705
Hip, Pigmented Villonodular Synovitis of 1170
Hip Shelves in Children. David M. Bosworth, J. William Fielding, William A. Liebler, Tadao Ishizuka, Hiroshi Ikeuchi, and Paul Cohen 1223
Hip, Suppurative Arthritis of 23
Histiocytosis 979
Histological Study of Rat Knee Joints 737
Homogentisic Acid 817

PAGE

Humeral Epiphysis, Fracture Separation of ... 333
Humerus, Deformity Following Supracondylar Fractures of the. Lyman Smith ... 235
Hypothenar Muscles, Duplication of, Simulating Soft-Tissue Tumor of the Hand. Report of a Case. Paul R. Lipscomb ... 1058
Hypothermia ... 945
Hypertrophic Changes in Cervical Vertebrae ... 392
Hypertrophy, Compensatory Muscle ... 278
Hypesthesia of Grafted Areas ... 261

I

Idiopathic Scoliosis ... 965
Ilium, Osteomyelitis of ... 23
Immobilization Atrophy ... 617
Immobilization, Effects of ... 31
Immobilization, Experimental ... 737
Impacted Fractures of the Femoral Neck ... 471
Implants, Metal ... 77
Infarcts, Idiopathic Bone ... 820
Inflammatory Response to Homogenous-Bone Transplant ... 811
Injuries to the Medial Ligaments of the Ankle. Result Study. O. Sherwin Staples ... 1287
Integrated Electromyographic Curve 1207
Intercorporeal Fusion of the Spine ... 1339
International Course of Traumatology and Orthopedy ... 1109
International Poliomyelitis Conference, Fifth ... 731
International Society of Orthopaedic Surgery and Traumatology ... 563, 731, 915
Interstitial Fluid, Blockage of Drainage of ... 609
Intertrochanteric Fracture ... 471
Intervertebral Disc, Ruptured ... 193, 565
Intervertebral-Disc Space in Children, Narrowing of the. Presumably an Infectious Lesion of the Disc. James R. Doyle ... 1191
Intervertebral Disc Space, Delayed Neurological Sequelae of a Retained Foreign Body (Lead Bullet) in the. Russell N. Shroyer, Charles H. Fortson, and C. B. Theodotou ... 595
Intervertebral Discs, Sequestrated ... 295
Intramedullary Fixation, Failure of ... 1201
Intramedullary Lipoma of Bone. Report of a Case. Martin C. Mueller and Jack L. Robbins ... 517
Intrinsic-Plus Deformity ... 951
Into-Talus Transposition of Tendons for Correction of Paralytic Valgus Foot after Poliomyelitis in Children. Anatol Axer ... 1119
Inversion Reflex of the Foot ... 859
***In Vivo* Osteocyte Death.** Harold M. Frost ... 138
Iodoacetate ... 165
Ionization of Metal Implants in Living Tissues, The. Albert B. Ferguson, Jr., Patrick G. Laing, and Edwin S. Hodge ... 77
Ischemia of Extremities, Prolonged Tourniquet. An Experimental Study on Dogs. Francis X. Paletta, Vallee Willman, and Arthur G. Ship ... 945
Isometric and Isotonic Contractions 1207
Italian Orthopaedic Association ... 368

J

Japanese Orthopaedic Association ... 730
Joint Changes Caused by Restriction 737
Joint Degeneration ... 31
Joint, Luschka's ... 853
Joint-Space Obliteration ... 1329

K

Kappa Delta Award ... 563, 731
Kinematics of Pisotriquetral Joint ... 1317
Knee, Adduction Injury of the, with Rupture of the Common Peroneal Nerve. Report of a Case. Max M. Novich ... 1372
Knee, Congenital Dislocation of the. John J. Niebauer and Don E. King ... 207
Knee, Deformity, in Gonadal Dysgenesis ... 600
Knee, Depressed Tibial Plateau Fractures of ... 783
Knee-Joint Compression ... 31
Knee Joint, Osteochondritis of the. A Report of Three Cases in One Family and a Discussion of the Etiology and Treatment. Alan DeForest Smith ... 289
Knee Joint, The Pathomechanics of the. I. The Medial Collateral Ligament and Lateral Tibial Plateau Fractures. Albert F. Martin ... 13
Knee Joints, Experimental Immobilization and Remobilization of Rat. [Correction, page 1108]. E. Burke Evans, G. W. N. Eggers, James K. Butler, and Johanna Blumel ... 737
Knee Mobilization in Hip Spicas ... 510
Knee, Transplanted Muscles about the ... 919
Knee, Painful ... 327
Kyphoscoliosis ... 1027

L

Lactic Dehydrogenase ... 457
Laminar Joints, Unilateral ... 1076
Late Appearance and Evolution of a Cortical Defect in a Boy with Delayed Puberty. Gerhard S. Schwarz ... 173
Legg-Calvé-Perthes Disease, Osteochondritis Dissecans Following. A Report of One Case. Alvin A. Freehafer ... 777
Length Discrepancy of Limbs ... 1

PAGE

Lepromatous Leprosy ... 661
Leprosy, the Hand in. A Seven-Year Clinical Study. Part I. General Aspects of Leprosy. Daniel C. Riordan ... 661
Leprosy, the Hand in. A Seven-Year Clinical Study. Part II. Orthopaedic Aspects of Leprosy. Daniel C. Riordan ... 683
Leprous Osteitis ... 683
Letter to the Editor. Gustav Rubin ... 727
Ligament, Injury to the Lateral Collateral, of the Knee ... 1372
Ligament, Medial Collateral ... 13
Ligament Tears in Tibial Plateau Fractures ... 783
Ligamentous Injuries of the Ankle ... 1184
Ligaments, Medial, of the Ankle ... 1287
Ligamentum Cervicis ... 311
Lipoma of Bone, Intramedullary. Report of a Case. Martin C. Mueller and Jack L. Robbins ... 517
Liposarcoma ... 1041
Low-Back Pain ... 193
Low-Back Pain in Association with Gout ... 341
Lumbar Disc Lesions, Correlation of the Myelogram with Clinical and Operative Findings in. W. Edward Lansche and Lee T. Ford ... 193
Lumbar Spine, Unilateral Laminar Joints of. Case Report. Malcolm D. Jones ... 1076
Lunate ... 1051
Lung Carcinoma ... 605
Luschka's Joint—A Degenerative Phenomenon. Caesar Orofino, Mary S. Sherman, and Daniel Schechter ... 853

M

Macroglobulinemia, Waldenström's ... 127
Magnesium Content in Bone ... 437
Malformation, Congenital, of the Atlanto-Axial Joint ... 1377
Malic Dehydrogenase ... 457
Malignant Bone Tumors in Siblings. Ewing's Tumor and an Unusual Tumor Perhaps a Variant of Ewing's Tumor. A Case Report. Robert W. Huntington, Jr., Donald J. Sheffel, Mortimer Iger, and Charles Henkelmann ... 1065
Malignant Degeneration, Unusual Case of Multiple Abnormalities of Bone with. Albert G. Smith, Ralph W. Coonrad, Delos W. Boyer, and Everett I. Bugg, Jr. ... 1329
Malignant Disease of Bone ... 127
Malignant Disease of the Extremities Treated by Exarticulation. Analysis of Two Hundred and Sixty-four Consecutive Cases with Survival Rates. James B. Troup and William H. Bickel ... 1041
Management of Fractures of the Greater Multangular. Report of Five Cases. Lee J. Cordrey and Miguel Ferrer-Torells ... 1111
Manifestations of Osgood-Schlatter's Disease in Late Teen Age and Early Adulthood. B. F. Woolfrey and E. F. Chandler ... 327
Medial Collateral Ligament ... 13
Membrane, Synovial, Changes ... 1170
Metabolic Disturbance in Osteochondritis ... 289
Metabolism, Bone ... 1349
Metabolism, Bone. IV. The Effect of Radioactive Strontium (Sr^{90}) on the Mobilization of Stable Strontium from the Skeleton of the Rat. Robert D. Ray and Irving Lyon ... 832
Metabolism of Muscle ... 617
Metabolism, Sulphate, in Cartilage ... 165
Metacarpophalangeal-Joint Instability ... 1015
Metal Implants ... 77, 1201
Metallic Fixation, Failure of ... 1201
Metaphyseal Fragmentation ... 407
Metastasis, Subperiosteal ... 1084
Microchemical Study of the Developing Epiphyseal Plate, A. Robert E. Kuhlman ... 457
Micropetrosis. Harold M. Frost ... 144
Mid-Central States Orthopaedic Society ... 365
Milkman's Disease ... 127
Mineralization of Bone Matrix ... 144
Mineralization, Skeletal ... 447
Mobilization of Stable Strontium ... 832
Monozygotic Male Twins, Coxa Plana in. Adolphus W. Dunn ... 178
Morphological Changes Consequent to Restriction of Motion ... 737
Morphology of Flexor Pollicis Longus 467
Motor-Unit Action-Potential Counts, Their Significance in Isometric and Isotonic Contractions. J. R. Close, E. D. Nickel, and F. N. Todd ... 1207
Multangular, Management of Fractures of the Greater. Report of Five Cases. Lee J. Cordrey and Miguel Ferrer-Torells ... 1111
Multiple Sclerosis ... 1165
Muscle, Action Potential of ... 59, 919, 1207
Muscle Anomaly ... 1058
Muscle Denervation ... 769
Muscle, the Electrical Response of Human Skeletal, to Passive Stretch. Robert O. Becker ... 1091
Muscle, Flexor Pollicis Longus. Its Morphology and Clinical Significance. Uberto Mangini ... 467
Muscle Imbalance, Correction of, in Cerebral Palsy ... 965
Muscle-Strength Evaluation ... 59, 919, 1207
Muscle as a Tissue (The Lankenau Hospital Conference) ... 561
Muscles, Experimental Excision of, in the Weanling Rat. Jonathan Cohen and Marvin S. Weinfeld ... 278
Muscles, Invertor and Evertor, of the Foot ... 1119
Muscles, Transplanted, about the

PAGE

Knee ... 919
Mycobacterium lepra ... 661, 683
Myelography ... 193
Myelography, Cervical ... 253
Myeloma ... 1041
Myeloma, Multiple ... 127
Myotomy, Adductor ... 111

N

Narrowing of the Intervertebral-Disc Space in Children. Presumably an Infectious Lesion of the Disc. James R. Doyle ... 1191
Navicular, Fracture of ... 1051
Navicular, Fracture of the Carpal. Diagnosis, Non-Operative Treatment, and Operative Treatment. Otto Russe ... 759
Necrosis, Avascular, of the Capitellum Humeri ... 513
Necrosis of Cartilage ... 31
Necrosis of Homogenous-Bone Transplants ... 811
Necrosis of Tendon Transplants ... 91
Neonatal Suppurative Arthritis ... 23
Nerve Graft ... 375
Nerve-Root Compression ... 392
Nerve, Rupture of the Peroneal ... 1372
Nerve Supply to Flexor Pollicis Longus ... 467
Neurectomy, Anterior Obturator ... 111
Neurofibrosarcoma ... 1041
Neurological Deficit ... 1027
Neurological Sequelae of Acute Trauma to the Spine ... 253
Neurological Sequelae, Delayed, of Retained Foreign Body in Intervertebral-Disc Space ... 565
Neuromuscular Impairment ... 945
New Bone Affection, A: Feathering. Harold M. Frost ... 447
New Jersey Orthopaedic Society ... 562
News Notes ... 365, 560, 730, 915, 1109, 1282
Nitrogen, Residual in Bone ... 1435
Non-Union of Fractured Navicular ... 759
North Carolina Orthopaedic Association ... 369
Nutrient Vascular System of the Tibia ... 626

O

Obituaries
Julius Hass ... 361
Norman Thomas Kirk ... 1450
William Benjamin McCracken ... 728
Duncan Clark McKeever ... 189
J. Warren White ... 359
Obturator Neurectomy for the Correction of Adduction Contracture of the Hip in Cerebral Palsy, Adductor Myotomy and. Henry H. Banks and William T. Green ... 111
Ochronosis ... 817
Odontoid Process, Hypoplastic ... 1337

PAGE

Oklahoma Orthopaedic Society ... 369
Olecranon ... 235
Opposition of the Thumb and Its Restoration. Bernard Jacobs and T. Campbell Thompson ... 1015
Orthopaedic Guild ... 368
Orthopaedic Research and Education Foundation ... 562
Orthopaedic Research Society ... 1457
Orthopaedist and Apical Lung Carcinoma, The. Paul E. Dee, R. Glenn Smith, Miles J. Gullickson, and Charles S. Ballinger ... 605
Osgood-Schlatter's Disease in Late Teen Age and Early Adulthood, Manifestations of. B. F. Woolfrey and E. F. Chandler ... 327
Ossification Center of the Capitellum 513
Ossification of the Posterior Aspect of the Elbow. Two Cases. Russell N. Shroyer ... 1156
Osteo-Arthritis Lipping ... 565
Osteo-Arthrosis Alkaptonurica ... 817
Osteoblasts in Tissue Culture, The Cytodifferentiation of. A Description of Cellular Emigrations from Embryo Chick-Leg Bones. George G. Rose and Thomas O. Shindler ... 485
Osteochondritis Dissecans Following Legg-Calvé-Perthes Disease. A Report of One Case. Alvin A. Freehafer 777
Osteochondritis Dissecans of the Talus ... 311
Osteochondritis of the Knee Joint. A Report of Three Cases in One Family and a Discussion of the Etiology and Treatment. Alan DeForest Smith ... 289
Osteochondrosis of the Distal Tibial Epiphysis. A Report of Two Cases. William L. Hassler, Clarence H. Heyman, and George W. Bennett ... 1261
Osteocyte Death, *In Vivo*. Harold M. Frost ... 138
Osteogenic Sarcoma ... 1041, 1329
Osteomalacic Bone ... 447
Osteomyelitis ... 565
Osteomyelitis, Acute Hematogenous. A Review of Sixty-six Cases. Jack L. Winters and Irvin Cahen ... 691
Osteomyelitis, Hematogenous ... 23
Osteomyelitis, Hematogenous Pyogenic Vertebral. Alexander Garcia, Jr., and S. Ashby Grantham ... 429
Osteomyelitis in Leprosy ... 683
Osteomyelitis, Pyogenic ... 414
Osteomyelitis of the Spine ... 414, 429, 1191
Osteophytic Displacement of Spinal Cord ... 392
Osteophytosis ... 853
Osteoporosis ... 127, 447, 817, 1349

P

Palmar Fascia ... 993
Pan American Medical Association ... 730
Pancoast Syndrome ... 605

PAGE

Panner's Disease ... 513
Paralytic Dislocation of the Hip ... 1223
Paralytic Valgus Foot ... 1119
Paraplegia ... 295
Patellar Tendon ... 1426
Pathogenesis of Dupuytren's Contracture, The. Experimental and Further Clinical Observations. Robert D. Larsen, Nahoto Takagishi, and Joseph L. Posch ... 993
Pathogenesis of Osteochondritis ... 289
Pathological Fractures of the Spine. Etiology and Diagnosis. A Review of One Hundred and Five Cases. James A. Nicholas, Philip D. Wilson, and Robert Freiberger ... 127
Pathomechanics of the Knee Joint, The. I. The Medial Collateral Ligament and Lateral Tibial Plateau Fractures. Albert F. Martin ... 13
Pauwels' Angle ... 471
Pedicle Flap, The Versatile Cross-Finger. A Report of Twenty-six Cases. H. Dean Hoskins ... 261
Pedicle Graft ... 375
Periarticular Ossification ... 1156
Perilunar Dislocation of the Carpus ... 1051
Periosteal Cells of Transplanted Bone, Survival of ... 811
Periosteal Hemorrhage ... 407
Periosteal Vascular System ... 626
Periostitis in Leprosy ... 683
Peroneal Nerve, Rupture of the ... 1372
Peroneal Paralysis ... 311
Peroneal Tendons, Clinical and Electromyographic Evaluation of Patients with Anterior Transposition of. William H. Blodgett and S. J. Houtz ... 59
Pes Cavus ... 311
Phase Conversion in Muscle Transplants ... 919
Phasic Activity of Muscle ... 59, 919, 1207
Philadelphia Regional Committee of Trauma of the American College of Surgeons ... 1109
Phosphoglucoisomerase ... 457
Pigmented Villonodular Synovitis with Invasion of Bone. Report of Six Cases. Paul E. McMaster ... 1170
Pisiform Bone and Pisotriquetral Joint, Roentgen Aspects of Injuries to the. Anthony Vasilas, R. Vincent Grieco, and Noel F. Bartone ... 1317
Plantar Dissection. An Operation to Release the Soft Tissues in Recurrent or Recalcitrant Talipes Equinovarus. Frederick C. Bost, Edwin R. Schottstaedt, and Loren J. Larsen ... 151
Plantaris Tendon Graft ... 1363
Plastics and Bone Repair [Editorial] 521
Poliomyelitic Muscles ... 1207
Poliomyelitic Patients, Electromyographic Study of Transplanted Muscles about the Knee in. David H. Sutherland, Frederic C. Bost, and Edwin R. Schottstaedt ... 919
Poliomyelitis ... 59, 919, 1119, 1207, 1223

PAGE

Polycythemia Vera ... 127
Polymorphous Dysplasia ... 1329
Polyurethane Polymer ... 521
Pott's Disease ... 295
Presidential Address. The American Academy of Orthopaedic Surgeons. Growth of the Vineyards. Harold A. Sofield ... 344
Presidential Address. The American Orthopaedic Association. The Closed Fracture of the Long Bones. John R. Moore ... 869
Primary Repair of Flexor Tendons. Claude E. Verdan ... 647
Problem of the Primary Curve, The. John R. Cobb ... 1413
Proceedings
American Academy for Cerebral Palsy 350
American Academy of Orthopaedic Surgeons, The. Scientific Sessions ... 875
American Academy of Orthopaedic Surgeons, The. Scientific Exhibits ... 892
American Medical Association: Section on Orthopaedic Surgery ... 1265
American Orthopaedic Association, The 1441
American Society for Surgery of the Hand ... 907
Association of Bone and Joint Surgeons ... 184, 1272
Forum on Fundamental Problems in Orthopaedic Surgery ... 538
Japanese Orthopaedic Association ... 541
Mid-Central States Orthopaedic Society ... 1270
New Jersey Orthopaedic Society ... 188
Orthopaedic Research Society, The ... 716
Piedmont Orthopaedic Society, The ... 353
Russell A. Hibbs Society, The ... 355, 1277
South Carolina Orthopaedic Association 720
Southern Medical Association. Section on Orthopaedic and Traumatic Surgery ... 722
Virginia State Orthopaedic Society, The 1105
Western Orthopedic Association ... 522
Profundus Tendon, Division of ... 1363
Prolonged Tourniquet Ischemia of Extremities. An Experimental Study on Dogs. Francis X. Paletta, Vallee Willman, and Arthur G. Ship ... 945
Prosthesis, Clinial Experiences with the S.A.C.H. Foot. Everett J. Gordon and Joseph Ardizzone ... 226
Pseudarthroses in Spine Fusions ... 1396
Pseudarthrosis, Congenital, of the Clavicle ... 337
Pyogenic Osteomyelitis and Arthritis of the Spine Treated with Combinations of Antibiotics and Gamma Globulin. A Preliminary Report. Burton A. Waisbren ... 414
Pyogenic Vertebral Osteomyelitis ... 429

R

Radioactive Strontium ... 832
Radioactive Sulphur in Chondrosarcomata. The Distribution, Concen-

PAGE

tration, and Effects of S^{35} after Intravenous Administration to Patients with Chondrosarcoma. Raymond G. Gottschalk ... 1239
Radius, Displacement of ... 333
Rare Subperiosteal Metastasis Eroding the Femoral Cortex Eight Years after Radical Mastectomy for Adenocarcinoma, A. Charles A. Borgia ... 1084
Reconstruction of the Distal Part of the Thumb after Traumatic Amputation. Restoration of Function and Sensation Using Nerve, Tendon, and Bone from the Amputated Portion. Carl E. Nemethi ... 375
Rectus Sheath, Anterior ... 993
Recurvatum, Congenital, of the Fibula ... 1258
Reflexes, Tonic, of the Foot ... 859
Remobilization of Immobilized Joints ... 737
Reticuloendotheliosis ... 979
Reticulum-Cell Sarcoma ... 1041, 1065
Rheumatoid Arthritis with Hypercorticism ... 127
Rib, Excision of ... 798
Rocker-Bottom Deformity ... 1119
Roentgen Aspects of Injuries to the Pisiform Bone and Pisotriquetral Joint. Anthony Vasilas, R. Vincent Grieco, and Noel F. Bartone ... 1317
Roentgenography of the Ankle ... 1184
Roentgenography, Stress ... 1287
Rotation of Vertebrae ... 1413
Rupture of the Medial Collateral Ligament ... 13

S

S.A.C.H. (Solid Ankle Cushion Heel) Prosthetic Foot ... 226
St. Louis Orthopedic Society ... 730
Sartorius Transplants ... 919
Sciatica ... 193
Sclerosis, Multiple ... 1165
Scoliosis ... 1413
Scoliosis, Attempted Treatment of, by Unilateral Vertebral Epiphyseal Arrest. H. R. McCarroll and William Costen ... 965
Scoliosis, Congenital ... 1027
Second International Course of Lyophilization ... 562
Sensory Testing ... 951
Septicemia ... 23
Serbian Medical Society ... 368
Sesamoid Bone ... 1317
Shoulder-Joint Disarticulation ... 1041
Shoulder, Painful ... 605
Simple Bone Cysts. Studies of Cyst Fluid in Six Cases with a Theory of Pathogenesis. Jonathan Cohen ... 609
Simple Night Splint for Correction of Tibial Torsion in Children. Rex J. Howard and L. P. Kleuser ... 177
Sinus Tarsi ... 311
Skin Graft, Split-Thickness ... 261

PAGE

Slipped Capital Femoral Epiphysis in Negroes. A Study of Ninety-five Cases. Caesar Orofino, John J. Innis, and Cedric W. Lowrey ... 1079
Society of Nuclear Medicine ... 732
Solid Ankle Cushion Heel Prosthetic Foot ... 226
Spastic Cerebral Palsy ... 111
Spinal Cord, Compressed, in Kyphoscoliotic Patients with Neurological Deficit, Transposition of the. With Special Reference to the Vascular Supply of the Cord. Richard C. Schneider ... 1027
Spinal-Cord Compression ... 392
Spinal-Cord Decompression ... 253
Spinal Involvement in Gout. A Case Report with Autopsy. Michael C. Hall and Golden Selin ... 341
Spine, Arthritis of ... 414
Spine, Cervical ... 253, 392, 565, 853, 1027
Spine, Cervical, Giant-Cell Tumor of ... 480
Spine, Curves of ... 1413
Spine, Destructive Lesion of, in Children ... 1191
Spine, Further Studies on Experimental Spondylitis and Intercorporeal Fusion of the. C. Roger Sullivan and Frank E. McCaslin, Jr. ... 1339
Spine Fusion, Anterior ... 295
Spine Fusion, Bone Growth after. A Clinical Survey. J. T. H. Johnson and Wayne O. Southwick ... 1396
Spine Fusion, Cervical ... 565
Spine, Lumbar, Unilateral Laminar Joints of ... 1076
Spine, Pathological Fractures of the. Etiology and Diagnosis. A Review of One Hundred and Five Cases. James A. Nicholas, Philip D. Wilson, and Robert Freiberger ... 127
Spine, Progressive Deformity of ... 965
Spine, Tuberculosis of ... 295
Splint, Night, for Tibial Torsion ... 177
Spondylitis, Experimental ... 1339
Spondylosis of the Cervical Spine with Compression of the Spinal Cord and Nerve Roots. Paul Teng ... 392
Stabilization of the Cervical Spine by Anterior Fusion. R. W. Bailey and C. E. Badgley ... 565
Stance-Phase Activity ... 919
Steinmann Pin, Failure by Corrosion of ... 1201
Steroidal Basis of Osteoporosis ... 1349
Strontium (Sr^{90}) ... 832
Strontium^{85} Metabolism in the Normal and the Castrated Mouse, A Study of. Lee J. Cordrey, James H. Christie, William J. MacIntyre, and Rufus Palmer ... 1349
Study of Experimental Trauma and Attempts to Stimulate Growth of the Lower Femoral Epiphysis in Rabbits, A. III. Lee T. Ford and Gregorio M. Canales ... 439
Study of the Residual Nitrogen in Bone Following Extraction with

PAGE

Ethylenediamine, A. Thomas E. Wheeler and George W. Hyatt 1435
Study of Strontium^{85} Metabolism in the Normal and the Castrated Mouse, A. Lee J. Cordrey, James H. Christie, William J. MacIntyre, and Rufus Palmer 1349
Subclavian Vessels, Compression of .. 798
Subluxation, Congenital, of the Knee 207
Sulphate Metabolism in Cartilage. II. S^{35}-Sulphate Uptake and Total Sulphate in Cartilage Slices. Rose Robyns Coelho and O. Donald Chrisman 165
Suppurative Arthritis of the Hip 23
Supracondylar Fractures of the Humerus 235
Surgery of the Hand in Cerebral Palsy and the Swan-Neck Deformity. Alfred B. Swanson 951
Swan-Neck Deformity 951
Syme Amputation 1
Synovial Fluid 31
Synovial Joint 853
Synovitis, Pigmented Villonodular, with Invasion of Bone. Report of Six Cases. Paul E. McMaster 1170
Syringomyelia, Confusion with Leprosy 661

T

Talar-Tilt Angle and the Fibular Collateral Ligaments, The. A Method for the Determination of Talar Tilt. [Correction, page 727.] Gustav Rubin and Morris Witten 311
Talipes Equinus Correction in Cerebral Palsy. Carl D. Martz 769
Talipes Equinovarus 151
Talofibular Ligaments 311
Talus, Lateral Dislocations of the ... 1287
Talus, Tilt of the 311
Talus, Transposition of Tendons into 1119
Tarsal Arthrodesis 769
Tarsal Varus 151
Tendon, Flexor, Ruptures in the Forearm and Hand. Joseph H. Boyes, James N. Wilson, and James W. Smith 637
Tendon Graft, Free, The Treatment of Profundus Division by. R. Guy Pulvertaft 1363
Tendon Grafts, Multiple 647
Tendon Reinsertion 375
Tendon Transplantation 151
Tendon Transplants 91
Tendon Transposition for Restoration of Thumb Opposition 1015
Tendons, Into-Talus Transposition of, for Correction of Paralytic Valgus Foot after Poliomyelitis in Children. Anatol Axer 1119
Tennessee State Orthopaedic Society 732
Tenolysis 647
Tenosynovitis, Granulatomatous, Due to Beryllium 1087
Tenosynovitis, Pigmented Villonodular 1170

PAGE

Tenotomies, Stirrup 769
Teratology Society, The 1109
Tetracycline Fluorescence in Bone Lesions. Preoperative Tetracycline Administration for the Identification of Abnormal Tissue during Operation and Tetracycline Concentration in Certain Lesions. John F. McLeay and Benedict R. Walske .. 940
Third Bi-annual Course on Modern Research Methods in Biology and Medicine 563
Thirteen Cases of Akkaptonuria from One Family Tree with Special Reference to Osteo-Arthrosis Alkaptonurica. Yasukazu Abe, Naozo Oshima, Ryoshin Hatanaka [correctly, Yoshiomi Hanakita], Tamikazu Amako, and Ryozo Hirohata 817
Thoracic Inlet, Tumors of 605
Thumb, Congenital Absence of the .. 658
Thumb, Opposition of the, and Its Restoration. Bernard Jacobs and T. Campbell Thompson 1015
Thumb-in-Palm Deformity 951
Thumb after Traumatic Amputation, Reconstruction of the Distal Part of the. Restoration of Function and Sensation Using Nerve, Tendon, and Bone from the Amputated Portion. Carl E. Nemethi 375
Thumb, Weakness of 1111
Tibia, Blood Supply of the Human. George E. Nelson, Jr., Patrick J. Kelly, Lowell F. A. Peterson, and Joseph M. Janes 625
Tibia, Congenital Absence of 1
Tibia, Dislocation of Proximal Portion 207
Tibia, Lipoma of 517
Tibial Bowing 1
Tibial Condyle, Medial, The Deformity of the, in Nineteen Cases of Gonadal Dysgenesis. Jerzy Kosowicz 600
Tibial Epiphysis, Osteochondrosis of the Distal. A Report of Two Cases. William L. Hassler, Clarence H. Heyman, and George W. Bennett 1261
Tibial Plateau 13
Tibial Plateau, Depressed Fractures of the. Treatment with Rigid Internal Fixation and Early Mobilization. A Preliminary Report. Charles Rombold 783
Tibial Torsion in Children, Simple Night Splint for Correction of. Rex J. Howard and L. P. Kleuser 177
Tibial-Tubercle Transplant in Children on the Growth of the Upper Tibial Epiphysis, The Effect of a. J. William Fielding, William A. Liebler, and A. Tambakis 1426
Tibial Tuberosity 327
Tibialis Anterior Tendon 151
Tibiofibular Fork 311
Tibiofibular Ligaments, Ruptures of 1287

PAGE

Toe-Grasping Reflex ... 859
Tonic Reflexes of the Foot. Their Orthopaedic Significance in Normal Children and in Children with Cerebral Palsy. William R. Duncan ... 859
Tourniquet Ischemia ... 945
Transaminase Activity ... 1008
Transcondylar (Diacondylar) Fractures of the Elbow ... 235
Transephiphyseal Bone Grafts ... 1381
"Transplantation" of the Compressed Spinal Cord ... 1027
Transplantation of the Index Finger in Congenital Absence of the Thumb. Eduardo Zancolli ... 658
Transplantation of the Tibial Tubercle ... 1426
Transplants, Heterogenous and Autogenous-Tendon. An Experimental Study of Preserved Bovine-Tendon Transplants in Dogs and Autogenous-Tendon Transplants in Dogs. J. Edward Flynn, John T. Wilson, Charles G. Child, III, and James H. Graham ... 91
Transposition of the Compressed Spinal Cord in Kyphoscoliotic Patients with Neurological Deficit. With Special Reference to the Vascular Supply of the Cord. Richard S. Schneider ... 1027
Transposition of Peroneal Tendons ... 59
Trauma, Esperimental, to the Femoral Epiphysis ... 439
Trauma, Unrecognized, in Infants and Children. Donald H. Altman and Richard I. Smith ... 407
Traumatic Dislocation of the Hip Joint in Children. A Report by the Scientific Research Committee of the Pennsylvania Orthopedic Society. John J. Gartland, *Chairman*, Albert B. Ferguson, Jr., William F. Donaldson, Marvin C. Goodman, Ervin E. Rodriguez, and Michael Skovron ... 705
Treatment of Profundus Division by Free Tendon Graft, The. R. Guy Pulvertaft ... 1363
Triquetrum ... 1317
Tri-State Orthopedic Society ... 732
Trochanteric Decubitus Ulcer ... 1165
Tuberculoid Leprosy ... 661
Tuberculosis of the Spine, Anterior Spine Fusion for the Treatment of. The Operative Findings and Results of Treatment in the First One Hundred Cases. A. R. Hodgson and Francis E. Stock ... 295
Tumor of Bone of Fibroblastic Origin 1143
Tumor, Giant-Cell, of the Cervical Spine ... 480
Turner's Syndrome ... 600

U

Ulcer, Trochanteric Decubitus ... 1165
Ulna, Displacement of ... 333
Ulnar-Nerve Transplantation ... 1156
Uncinate Processes ... 853
Unilateral Laminar Joints of Lumbar Spine. Case Report. Malcolm D. Jones ... 1076
Unrecognized Trauma in Infants and Children. Donald H. Altman and Richard I. Smith ... 407
Unusual Case of Multiple Abnormalities of Bone with Malignant Degeneration. Albert G. Smith, Ralph W. Coonrad, Delos W. Boyer, and Everett I. Bugg, Jr. ... 1329
Uric Acid ... 817

V

Valgus Foot, Paralytic ... 1119
Varus Deformity of the Elbow ... 235
Vascular Compression of Spinal Cord 392
Vascular Patterns in Immobilized, Denervated, or Devascularized Rabbit Limbs. Albert B. Ferguson, Jr., and Yoshihiko Akahoshi ... 617
Vascular Supply of Spinal Cord ... 1027
Vascularization of Autogenous and Homogenous-Bone Transplants, Comparative. B. Leslie Hammack and W. F. Enneking ... 811
Versatile Cross-Finger Pedicle Flap, The. A Report of Twenty-six Cases. H. Dean Hoskins ... 261
Vertebral-Body Fractures ... 127
Vertebral Collapse ... 127
Vertebral Epiphyseal Arrest ... 965
Vertebral Growth, Measurement of ... 1396
Vertebral Osteomyelitis ... 414, 429
Villonodular Synovitis, Pigmented ... 1170
Volar Transnavicular Perilunar Dislocation of the Carpus. Alexander P. Aitken and Edward A. Nalebuff ... 1051

W

Walking Patterns ... 59
Water Content of Muscle ... 1008
Wedging of Vertebrae ... 1413
Weight of Muscles ... 278
Well Differentiated Fibrosarcoma of the Calcaneus. Report of a Case Treated by Resection. Raphael R. Goldenberg ... 1151
Western Orthopedic Association ... 730
Wrist, Flexion Deformity of ... 951
Wrist, Injury to ... 1111
Wrist, Mid-Carpal Fracture-Dislocation of the ... 759

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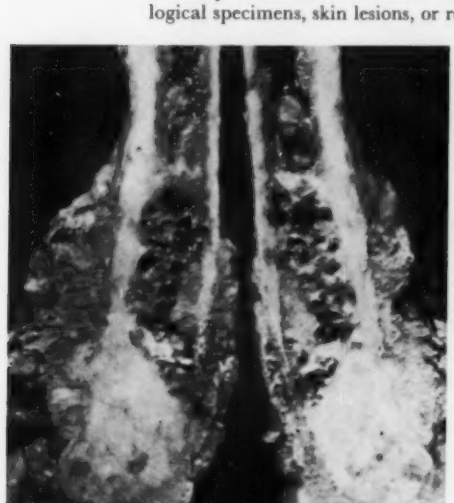

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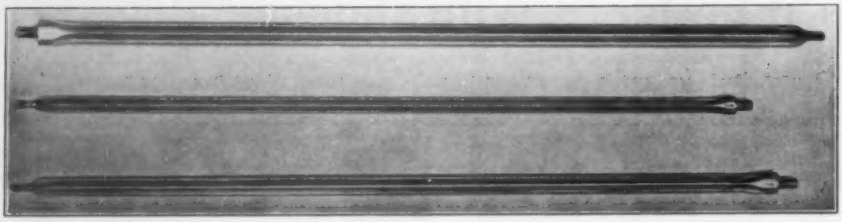

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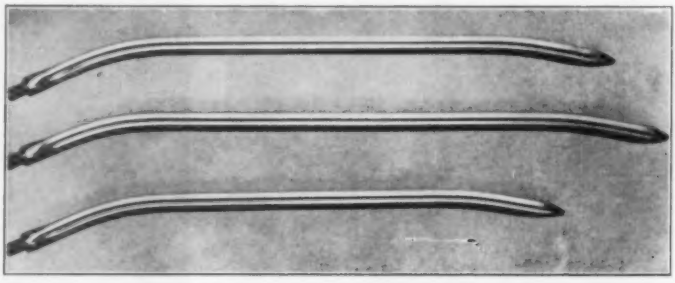

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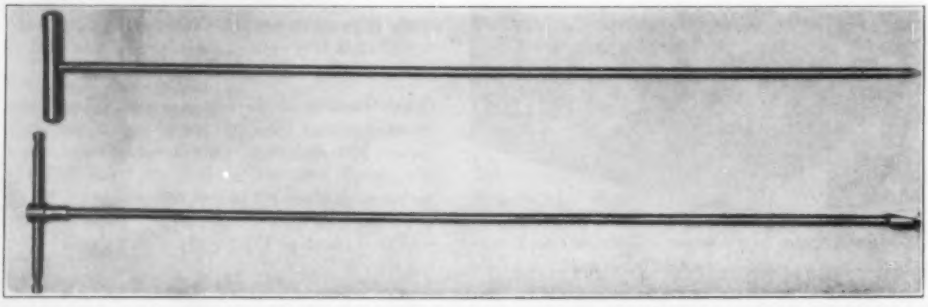

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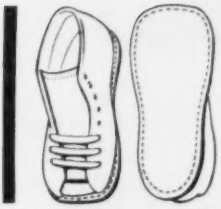

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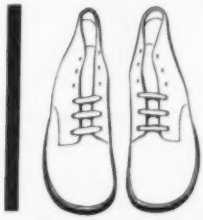

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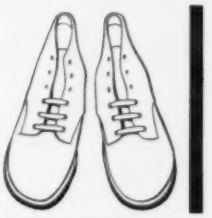

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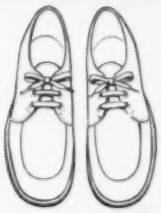

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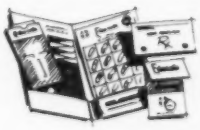

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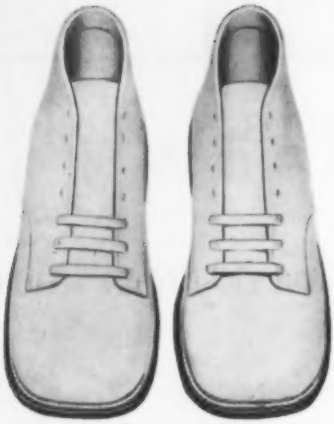

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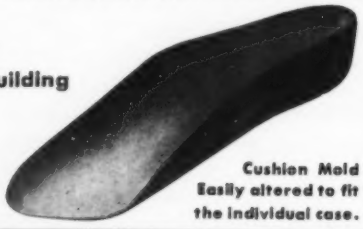

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LIST OF ADVERTISERS — *December 1960*

	Page
Acme Cotton Products Co., Inc.	39
Aeroplast Corporation	34
Aireloom Bedding Company	14
C. H. Alden Shoe Company	32
American Association for the Advancement of Science	54
American Sterilizer Company	17
Austenal, Inc.	26, 27
Bauer & Black, Division of the Kendall Company	8, 9
The Berivon Company	35, 52
Bowen & Company, Inc.	30
S. H. Camp and Company	46
G. W. Chesbrough Co.	21
Chesebrough-Pond's, Inc.	37
Codman & Shurtleff, Inc.	23
DePuy Manufacturing Company, Inc.	16
Eastman Kodak Company	43
J. Edwards Shoe Company	49
Elgin Exercise Appliance Co.	56
Florida Brace Corporation	40
Florida Manufacturing Corporation	58
Freeman Manufacturing Company	29
General Electric Co., X-Ray Dept.	11
S. R. Gittens	50
Hack Shoe Company	47
J. E. Hanger, Inc.	48
Herbst Shoe Manufacturing Company	25
House of Bidwell, Inc.	57
Kleistone Rubber Company	56
Lederle Laboratories, Division of American Cyanamid Company	5, 6
Lindell Pattern Works	50
McNeil Laboratories, Inc.	19
M. J. Markell Shoe Company, Inc.	18
Medic Shoe Manufacturers, Inc.	50
Medical Research Specialties	20
Merck Sharp & Dohme, Division of Merck & Co., Inc.	15
Miller Brace Co.	56
Minnesota Orthopedic Services	57
The C. V. Mosby Company	55
V. Mueller & Co.	41
New York University—Bellevue Medical Center	52
Orthopedic Equipment Co.	4
Orthopedic Frame Company	33
Philadelphia Regional Committee on Trauma	54
J. A. Preston Corp.	57
John M. Richards, M.D.	54
Richards Manufacturing Company	28, 51
I. Sabel, Inc.	7
Chas. A. Schmidt Surgical Instrument Company	44
Spencer, Incorporated	45
Charles C. Thomas	53
Truform Anatomical Supports	38
Wallace Laboratories, Division of Carter Products, Inc.	12, 13, 22, 31
Wallace Orthopedic Manufacturing Co.	10
Wright Manufacturing Company	36
Zimmer Manufacturing Company	24, 42

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